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ORIGINAL ARTICLES

PRESIDENTIAL ADDRESS: OBSTETRICAL RESULTS*

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LADIES AND GENTLEMEN:

You have honored me beyond my merit with the highest office in this association, and for the past year and a half it has been my humble pleasure to serve to my utmost ability your best interests in promoting the ideals and traditions of the Southern Minnesota Medical Association. This organization has no axes to grind but is wholly interested in bringing together, once a year, all members of the association and their friends, regardless of geographic boundaries. Primarily, its object is to increase knowledge and skill in the healing art, to build up a more ethical and tolerant profession and emphasize, most of all, the needs of the general practitioner.

I wish to quote Dr. Hugh J. Means, speaking before the Columbus (Ohio) Academy of Medicine, voicing a thought that is now quite firmly entrenched in the minds of practitioners all over the country, when he said: "The cults always have been, are and always will be with us. Too much effort is expended in attempting to suppress the fact of the day and not enough in searching for the true cause that enables it to exist. Physicians, as a whole, pay too little attention to the psychology of medical practice and consider medicine as an exact science, forgetting that the patient is an individual with prejudices, idiosyncrasies and temperaments. A keen analytical mind in ordinary affairs will function in an absurdly illogical manner in health matters. When a patient consults a physician, his mind is made up in advance that there is something wrong. When told that his illness is merely a state of mind or something of small moment, he may appear satisfied; but deep in his heart he is disappointed. We all rather enjoy the opportunity to excite a little sympathy for ourselves and after recovery like to think that our illness was a little more severe than ordinary. The patient wants something done to him. The less serious his condition, the more the process is enjoyed; not being satisfied merely with advice, which to his unscientific mind is more or less intangible, the patient drifts to a healer. It may be generally admitted that physicians themselves are responsible to a large degree for the drift to cults of persons who complain of being ill, when no definite pathology is present, because of a disposition to dismiss the patient with the admonition that "there is nothing wrong."

Let us say that one-third of the patients who consult a physician are scarcely in need of medical attention, as far as real demonstrable ailments are concerned. These patients are entitled to careful, thorough examination and attention and not the usual cursory hurried examination with a little indefinite advice regarding habits and possibly a few recommendations as to eating and exercise. Thus, patients pass from one physician to another and, receiving the same attention and advice, gradually become more and more dissatisfied. This is the main reason for quack cults, practices and all manner of irregulars. If a patient leaves the office unsatisfied, it is usually the fault of the physician. We have never heard of a patient consulting an irregular and being dismissed without the recommendation of a complete course of treatment and it would be difficult to estimate how many millions of dollars fall into the hands of the unqualified, the unscrupulous and the rank unethical.

This problem is worthy of the most serious consideration. How are we to treat these patients? There can be but one answer. We must give this type of patient the time and serious attention he deserves or he will continue to encourage and patronize the ignorant and unqualified. Hygienic instruction and sensible advice are all available and distinctly helpful, but the average patient is not

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satisfied with this. He or she, especially she, wants some kind of active treatment upon which hope and confidence can be based. Because these patients will probably recover without active treatment is no good reason for relying entirely upon the unaided forces of nature. Many of them will be satisfied with this and will need no further attention; others will require helpful direction and re-education, even medication, though its effect is more mental than physical in character.

In order to combat the cultist, the practitioner will find it to his advantage to at least know on what his practice is founded. He will find, very likely, that regular medicine can give every real advantage offered by the special cult and more. The patient can be advised by his physician as to diet, water intake, exercise, relaxation and even as to attitude of mind. "The doctor of today has greatly neglected his psychology and has given to the mental healer an unusual opportunity," says Dr. Hubbard of the New York Board of Health. "If the physician does not intellectually respect the complex matters of the soul and their interdependence," to quote the late Dr. Wier Mitchell of Philadelphia, "he is unfit for higher seats in the temple of healing. Through closely studying and appraising his enemy, the unqualified practitioner, and especially keeping in touch with the latest developments in both medical and mental science, the physician of the legally recognized schools can successfully cope with the menace of the cults."

Great advancement in medicine during the past twenty-five years has been accomplished and a few of the more important discoveries may be briefly mentioned.

Major Walter Reed and James Carroll in 1899 proved the cause of yellow fever to be either an ultramicroscopic organism or a filterable virus transmitted to man by a particular species of mosquoto, the stegomyia fasciata.

A quarter of a century ago, our teachers talked of the etiology of lues. The *spirocheta pallida* was unknown. In May, 1905, Fritz Schandinn, working with Erick Hoffmann, crowned his life work by the discovery of *spirocheta pallida.* This was the first step in the conquest of syphilis. The next was the biochemical discovery in 1910 by Paul Ehrlich of Strehlen, Silesia, of salvarsan, and his work enabled August von Wassermann in 1916 to hit upon the specific and extremely reliable diagnostic test for syphilis.

One of the greatest medical achievements in the World War was the conquest of wound infection, through the introduction in 1915 by Carrel and Dakin of a physico-chemical principle of wound irrigation by a gas in solution.

A student senior of the University of Minnesota in 1899 was asked in a quiz class of the late Dr. Alexander Stone how he would diagnose a stone in the ureter. To the consternation and amusement of the class, he replied: "I would pass a probe into the ureter." The professor replied: "Young man, you will be dead and resurrected a thousand years before you will ever pass a probe into the ureter"; but the introduction of the cystoscope by Max Nitze in 1916 vastly improved the surgery of the bladder and ureter.

The discovery of insulin by Banting and Best marks an epoch in the treatment of diabetes, the previous treatment of which had been so unsuccessful and discouraging. This remedy will prove invaluable in prolonging and saving valuable lives. In time I hope there will come a demand for a course of instruction in our public schools in dietetics which might serve to check the apparent increase in this prevalent disease.

One of the reasons why physicians in general have failed to hold or to satisfy their patients is the fact that no record is kept of their cases; were this done and case records reviewed and studied it would prevent repetition of mistakes in diagnosis and treatment and aid in cleaning up the cult problem. This, of course, takes time, but it pays to do it.

Our records of obstetrical patients, kept for twenty-five years in a country and city practice which totaled 1,032 cases, very few of whom were hospitalized, serve as an illustration. The anesthetic was chloroform; gas or ether was used in a few cases. No untoward effects were observed and I would say that chloroform, intelligently and properly administered, is a very satisfactory anesthetic in obstetrical work. Gas tanks are too cumbersome to carry and its administration is impractical in that assistants are, as a rule, few and untrained and the physician must perform the added duty of nurse and anesthetist. When the labor pains are sufficient to insure continuance, the anesthetic is administrated in amount to relieve the sharp edge of the pain. At the completion of the second stage, the anesthetic is pushed to primary

anesthesia and seldom, if ever, do I have a case of nausea or vomiting following delivery.

As to abnormal presentations and complications of pregnancy, we note the following:

Post partum hemorrhage. — Fourteen cases in number. Most of these cases were controlled by the usual measures such as kneading the uterus and the administration of ergot and pituitrin; but a few cases required a pack for several hours. Holding and firmly manipulating the fundus usually will prevent and control hemorrhage, except in uterine inertia or laceration of cervical vessels.

Contracted pelvis.—Ten cases. Seven of these, all in the same mother, were very difficult, required forceps, and resulted in the loss of three of her infants from pressure and asphyxiation. The last and most difficult of all, was a breech presentation. Failing in external version, the aftercoming head was finally delivered with forceps. The eighth case, a primipara seen too late to remove to a hospital, was delivered, with the assistance of two other physicians, by forceps, resulting in the child's death due to pressure and asphyxiation. The mother was advised in the future, should the occasion arise, to go to a hospital for cesarian section. This she failed to do and, in the hands of another physician, delivery resulted fatally to the child. Her third delivery, an operation at Rochester, resulted favorably to mother and child. This demonstrates the importance of careful repeated examinations and watchfulness to detect complications in advance, so as to be prepared for any emergency. The ninth case of contracted pelvis was in a primipara, upon whom a cesarian section was performed successfully. The mother, a minor, lived to appear in court as a witness against her seducer, who was duly sentenced to serve a term in the State Penitentiary.

Adherent placenta.—Seven cases. All required manual removal, but with uneventful recovery.

Twins.—Seventeen cases with two infantile deaths. One case of triplets was ushered into the world. The diagnosis was twins. The first infant, a female, weighed three pounds and was born 4:15 a. m. This satisfied the fond father, who had previously notified me that future calls would not be forthcoming if the prospective was not a girl. But complications arose and the second child, a female, weight three and one-half pounds, was born at 5 a. m. The third child, a female, weight two and one-half pounds, was born at 5:10 a. m. This was more than the father had figured on and I assured him that he was abundantly supplied with girls, to which he replied: "Well, I did not want three." The mother and children are doing well.

Breech presentations.—Thirty cases. Two of these cases were high, so that a hook was used around the groin over the thigh, resulting in perforations of the abdominal wall at Poupart's ligament in both cases. The wounds under treatment healed quickly. One case with prolapsed cord was delivered successfully, but another with premature rupture of membrane was very difficult. One must have a fully dilated cervix for delivery of the aftercoming head, the danger being that the aftercoming head will be caught by the insufficiently dilated cervix and unless the child is delivered in from five to seven minutes from the time of the appearance of the cord at the vulva, death may result. The extraction of the aftercoming head in this case required forceps, the resulting trauma and traction causing its death the following day.

Stillbirths. — Twenty-eight cases. A version was performed in one case of malposition, but on account of the extreme size of the aftercoming head, craniotomy was necessary. Of the remaining, one was a monstrosity; one, a dry labor breech presentation; four, contracted pelves; one, strangulation by the cord before my arrival; one, strangulation with loops of cord in forceps; and the rest to causes such as prematurity, lues and asphyxiation by prolonged labor.

Dry labors.—Thirty-four cases. Rupture of membranes prematurely ranging from a few hours to seven days.

Version.—Six cases. One for pendulous abdomen and non-engagement of head; one, placenta marginalis; one, placenta abruptio; and the rest for causes already mentioned.

Eclampsia.—Three cases. One, postpartum, made a good recovery; one, antepartum and postpartum with recovery of mother and child; and one in which albuminuric retinitis forced induction of labor at eight months, with uneventful recovery.

Posterior positions. — Nine cases with one death from version.

Face presentations.—Nine cases.

Placenta marginalis.—Four cases.

Placenta centralis.—One case, eight months parturition, no fatalities.

Acute appendicitis.—One case. Abscess was drained at hospital, seven days postpartum, with good recovery.

Uremia.—One case, ten days postpartum, with maternal death.

Bartholin gland abscess.—Two cases.

Deformed and crippled cases.—Twelve in number. One, a bifid thumb at the second joint, was operated under local anesthesia while the child slept; one case, absence of left forearm and hand; two cases each with twelve toes and twelve fingers, which supernumeries were amputated at birth with excellent result; club feet, four cases; one infantile hand with web fingers to the second joints.

Spina bifida.—Three cases. One, located in the cervical region, was operated in the city about the second month with good recovery; the other two, in the lumbar region, the thin membranous type, became infected, resulting fatally.

Hydrocephalus.—One case, which died.

Two interesting cases should be mentioned: One was a child with cranial tabes; rectal palpation gave the impression of a breech presentation; delivery was slow on account of sluggish uterine contractions. The soft head presented a large hernia of the vertex, which was due to the absence of bony structure of parts of the parietal and temporal bones. Strange to say, this filled in with bony growth in a few months. The other case, of lesser involvement about the posterior fontanelle, looked like an ordinary caput succedenium, but was really a hernia of the brain, 7.5 to 10 cm. in diameter. This gradually disappeared with complete ossification in three months.

Ectopic gestation.—Two cases. One was sent to the hospital with recovery from operation and one died before she could be taken to the hospital. This was about twenty-five

years ago. With modern good roads and auto ambulance, we most likely could have saved her.

Two mothers died of pulmonary tuberculosis shortly after childbirth and one with influenza died suddenly in the second stage of labor.

Forceps cases.—One hundred and sixteen. The large majority were normal cases in which advance was delayed in the parturient canal or a long or dry labor threatened to exhaust the mother, endanger the child by asphyxiation or give rise to hemorrhage from uterine inertia. Forceps are a very great help when indicated, but one should always study the indications carefully and wisely before applying them, as the normal delivery is the safest to mother and child. How many times have we thought we would need forceps and have had them boiling to find that the child was born before we could use them.

Pituitrin is a valuable aid only where wisely used. With an undilated cervix or a posterior high position, to give pituitrin invites disaster. These cases are best treated by correcting the posterior position, or ironing out the rigidos with the sterile gloved fingers, by performing a version or applying forceps. In this way a ruptured uterus or a dead child will be averted.

The late Dr. Merritt, a most excellent obstetrician, informed me that he had never applied forceps to a living child without bringing it alive into this world. This requires great skill, experience and good judgment, and one should well strive to attain this proficiency.

INSULIN AND KETOSIS

Ketosis is not confined solely to cases of diabetes. It is an accompaniment of carbohydrate starvation, however produced. Acidosis is not infrequently found in preoperative or postoperative conditions, owing to enforced deprivation of food for one reason or another. It is found accompanying so-called toxic vomiting, sometimes particularly in the persistent type seen in pregnancy. Attention has been called to the use of insulin in the treatment of nondiabetic acidosis. The alleviation of this condition through the administration of glucose by rectum or parenterally has been demonstrated. More prompt success has been reported through the combined use of insulin hypodermically and glucose intravenously. While the treatment gives promise, it is not free from danger. Insulin therapy demands care in the case of a diabetic patient. Doubly great is the need of intelligent precaution with the nondiabetic patient.—*Jour. A. M. A., May 24, 1924, p. 1695.*

RADIUM AND RADIUM EMANATION

Radium is commonly supplied in the form of radium salts enclosed in containers. Also, tubes containing radium emanation are available. Salts of radium disintegrate at a rate so that 1,780 years elapse before the compound loses one-half of the initial activity. On the other hand, radium emanation loses about 0.75 per cent of its activity each hour, and consequently its activity is practically gone after one month.—*Jour. A. M. A., May 3, 1924, p. 1462.*

RECONSTRUCTIVE SURGERY AND REHABILITATION—THEIR RELATION TO SURGERY OF TRAUMA*

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In the field of general surgery and in the special fields of orthopedic and plastic surgery are found many deforming conditions which, uncorrected, handicap the individuals, rendering them unfit to compete in employment and to enjoy the usual pursuits of the average able-bodied man. During the war many old metholds were revived and new ones invented which aimed at the prevention or correction of physical deformities. This work was classified as physical reconstruction and the necessary surgery used to overcome the disability was called Reconstructive Surgery. This type of surgery combines with the necessary surgical procedures such adjuncts as physiotherapy, occupational therapy, retraining exercises, and a continuous follow-up or supervision of the case, by the surgeon, until as complete a functional restoration as possible is obtained.

Many of these handicapped individuals are so badly disabled that function cannot be restored to the point of permitting return to their old occupation, therefore, hand in hand with the developments in medicine and surgery resulting in consideration of reconstructive measures, has been born an economic development known as Rehabilitation of the Disabled. Rehabilitation deals with those measures necessary to make a disabled individual fit to resume his economic position in society. Rehabilitation of the disabled embodies five logical steps, often overlapping in function, but all aiming at the final economic end-result, independency.

These steps are:

1. Physical and mental restoration, by adequate and completed medical, surgical and hospital care;
2. Convalescence, under environment which tends to retrain work habits, to harden and strengthen the individual rather than under environment which tends to hospitalize, parasitize and make possible the development of functional neuroses;

*Presented before St. Paul Clinic Week, St. Paul, January, 1924.

3. Retraining, when necessary, for a new occupation, or an occupation allied to his old work—called vocational training;
4. Replacement at suitable employment;
5. Continued medical and economic supervision to see that his rehabilitation is completed and so remains.

Prior to the war examples of rehabilitating the disabled were found chiefly in the work of the medical departments of a few great industries. The return of their permanently disabled employees, following work accidents, to new types of occupations which they could carry on and which usually had some incentive for advancement; the selected occupations for their cardiac cases; the treatment in sanatoria of their tuberculous employees and, when the disease was arrested, their return to selected, suitable occupations in the plant, all furnish excellent examples of the meaning of rehabilitation. Unfortunately only a very small percentage of the total disabled population of the nation was included in this number. Nevertheless this pioneer work paved the way for this new medico-economic movement.

During the war the medical department of the army developed great plans for the Physical Reconstruction and Rehabilitation of the Disabled Soldiers. Suffice it to say that prior to the wholesale discharge of these disabled soldiers following the armistice, this work of salvaging these men was progressing exceedingly well. It was during these months that the foundation was laid for the present scope of the rehabilitation movement. Time and again it was pointed out that the casualties from the great industrial army in this country were annually more than three times as great as the casualties in our military army for the entire war. Yet no national movement for the reclaiming of these disabled civilians had ever been launched.

As a result of this propaganda New Jersey in 1919 passed the first state law for the Vocational Rehabilitation of the Disabled from Industry. Since then thirty-five other states and the Federal government have passed similar laws—some of them broad enough to include the disabled from disease, accident or congenital deformity. These laws all provide for the vocational training of these disabled, the furnishing of artificial appliances when necessary and the replacement at suitable employment; the funds for this service being furnished by equal appropriations from the state and federal governments. Five of the states function without federal aid. The laws are broad enough to include physical restoration, and are so interpreted in a few states, for they read: "To render a disabled person *fit* for remunerative employment."

All working in this field now realize that a great many of the cases can be improved physically, and must be so improved, before their vocational training and their economic rehabilitation can be assured. Thus reconstructive surgery, or medical reconstruction, is often the connecting link between the disabled and his future remunerative occupation.

Estimates differ as to the number of disabled in each state entitled to the benefits of the rehabilitation act. Thus, in Illinois, one group has reported that approximately 1,000 persons annually will be found needing this service, while another group claims that over 100,000 permanently disabled men and women can be found coming under the act. Remember that the total number of disabled now in our midst are being added to annually by the increasing number of industrial, automobile and public utility accidents. Statistics quoted by me* showed that annually 875,000 people were disabled for more than four weeks by industrial accidents alone; that 76,000 suffered loss of members; that 200,000 were otherwise permanently disabled and that 28,000 of our people were killed in industry. Statistics quoted by other writers since then show even a higher casualty list. This does not include the thousands who are disabled on our streets, in the homes, on the farms; neither does it include the disabled from occupational diseases, the cardiacs, the tuberculous, the paralytics, the mental defectives and others disabled by disease and congenital defects. All of these cases, if above the working age, are entitled to rehabilitation by the states having such laws. The great majority require some form of physical reconstruction to *fit* them for remunerative occupation. These facts give one some conception of the size of the medical and surgical problem involved.

Every paper or address should have some real motive for its publication or delivery. Lest you miss the objects of this paper, I want to insert here the two points I am endeavoring to drive home:

1. To increase the knowledge of our profession concerning these rehabilitation laws, and the im-

*Reconstructive Surgery, Minnesota Medicine, June, 1921.

portance of every physician referring his permanently disabled patients to the proper authorities so that they may receive this great opportunity of economic independence.

2. To urge the early application of reconstructive measures to every serious injury case, with the view of preventing permanent disabilities as far as possible, and of preventing traumatic neuroses and habits of idleness and dependency which frequently are more disabling than real deformities.

It is surprising how few of our surgeons, and our hospitals, have any real knowledge concerning rehabilitation or the fact that their permanently disabled patients can be referred to the state rehabilitation commission for retraining and placement at useful occupations. They are so in the habit of thinking only of treating the injury until healing has occurred that their minds do not function beyond that stage into the field of the patient's future usefulness. Or, if they do look into his future it is only with a sense of sorrow and pity for another victim of accident or disease.

Recently I witnessed the amputation of a leg by one of our best surgeons. The patient was a comparatively young man, a laborer, with a family. The surgeon said: "Poor fellow, he won't be able to go back to his usual work; he knows no other work; I wonder what will become of him and his family." This surgeon was surprised and gratified to learn that his patient could be referred to a state department where an artificial leg would be furnished him and where he might be trained for a new job.

In this case the amputation was done because of a disease not related in any way to his work; therefore, this patient was without employee's compensation. In our city, the charities or some other philanthropic organization can be found, which would finance this family during the period of the father's retraining for a new job. As yet the state law does not provide for maintenance during the period of training, and it is questionable whether this is advisable. The majority of these cases, however, can be trained for new work in some industry where they will receive some wage during training. In our community, workshops exist and others are being provided where the more serious cases can receive training and at the same time be paid wages, the money for this being obtained by the sale of fabricated articles plus one-half the weekly sum the charities would otherwise have to provide for the maintenance of this family. The sum from the charities is paid direct to the workshop management rather than to the individual—the latter is thus unaware of the fact that he is receiving aid.

The following case which I have had under my care for the past two years illustrates how many of these cases may be handled:

F. K., aged 34, married, two children, lived in a small rented flat, employed as a fireman and engineer in a large office building. In February, 1922, an elevator which he was operating fell seven stories. Patient was brought direct to my service at St. Luke's Hospital suffering from backward dislocation of right knee, lateral dislocation of right patella, fracture and complete outward rotation of external condyle, fracture and complete displacement of internal condyle into knee joint cavity.

Treatment consisted of immobilization and traction in a Thomas splint with fomentations to joint for one week; reduction of dislocation under gas anesthesia with continued immobilization and traction in Thomas splint; operation two weeks later on external condyle, gas anesthesia, consisting of replacement of fragment and fastening of same to shaft of femur by ivory peg (I now use beef bone pegs); one week later, operation on internal condyle, gas anesthesia, replacing fragment in normal position as nearly as possible and holding same in position by drilling two holes each through shaft and fragment, threading kangaroo gut through these and tying in place.

This last operation was indicated because this internal condyle lay in such a position that weight bearing would be between the head of the tibia and this pointed fragment with ankylosis of knee bound to result.

This last operative wound became infected—staphylococcus albus. Bone developed in the capsule and tendons about the joint and even in the muscles—a condition similar to myositis ossificans. Two subsequent operations for drainage and removal of necrotic bone were necessary. A small discharging sinus has persisted in this case but is gradually healing.

Ordinarily this patient would be continued on weekly compensation of $16.00 per week by the industrial commission until this wound had completely healed and then a settlement would be arranged based on the percentage of total disability. The healing of this wound may take two or three years. By the end of that time this patient would

be a confirmed loafer and pensioner. Too often have I seen the pitiful mental attitude of these long drawn out chronic cases.

During this patient's stay in the hospital he was given various forms of occupational therapy. Soon it developed that he had a natural inclination toward mechanics. This was fostered by talks with the patient, and by his reading, and ward occupations having a vocational trend. One year ago, when it was felt that patient could go about, returning twice or three times a week for dressings, the following arrangement was made:

The insurance company settled with patient for a loss of 25 per cent of his leg. Settlement at this time on existing condition would have been a 50 per cent loss, but I assured patient and the insurance company that this loss would be decreased one-half by time and further treatment. Patient also had eighteen months from time of this settlement to re-open case if my predictions of improvement failed. The insurance company guaranteed all further surgical care needed. This particular insurance company has grasped the ideals of rehabilitation.

Patient was then referred by me to the representative of the state rehabilitation commission with my recommendations for training in a practical course of mechanical engineering. This was arranged and patient was sent to the Armour Institute. After a short time he was given a part time job in the engineering department at Armour's plant and the remainder of the day was spent at the Institute. At present he is continuing his studies by practical full time employment at Armour's. Recently he was transferred to the draughting department. He received wages from the time he started at part time employment. At present he receives $34.00 per week although he is still in training. The lump sum settlement he received from the insurance company was used as a part payment on a duplex apartment. Patient and his family live in one apartment and rent the other. He is keeping up his payments and making money in addition on his rent savings.

Recently this patient told me that his accident was a lucky thing for him. Before it occurred he was a satisfied laborer but now he is learning a good trade, owns his home and is making more money than he ever made before. This is the ideal of compensation expressed in terms of rehabilitation—not merely a pension over an indefinite period, but a future compensation for the injuries sustained expressed in terms of a happier, more contented, more productive member of society

Turning now to the second point in this paper, namely the application of reconstructive methods to all traumatic cases with a view of preventing or diminishing permanent deformities, traumatic neuroses and habits of idleness and dependency.

Recently a representative of a large casualty insurance company approached me with the proposition of doing their reconstructive surgery on their bad results from industrial surgery. My reply was that if they would do good reconstructive surgery from the beginning of the injury they would have fewer bad results and less need for a surgeon to do corrective work after the damage was done. Gradually most of our representative casualty companies are learning the lesson that the more expensive surgery in the first place is the cheapest in the long run.

During the last year there have been referred to me for reconstructive cure, that is, restoration of function, eight fractures in or near the shoulder joint, twenty-one cases of periarticular shoulder injuries, of from one month to three years' standing, including subdeltoid and subacrominal bursitis, traumatic arthritis or periarthritis, tearing off of the greater tuberosity, tendosynovitis of the long head of the biceps, et cetera, a constricting scar and an abscess of the shoulder joint.

In practically every one of these cases a careful review or backward look revealed the time or place when certain reconstructive methods if properly applied would have prevented or greatly reduced these permanent disabilities. The early and frequent use of the *x*-ray; the application of splints or traction in such a way as to prevent the stiff joints or the useless arm hanging at the side; the early use of physiotherapy; the developing of work habits early rather than waiting until months of hospital care was completed, are all methods of reconstruction which should have been applied as continuous treatment from the onset of the injuries instead of waiting until months later when the cases were referred for reconstructive surgery.

Reconstructive methods involve not only the proper diagnosis and proper early treatment of the injury, but certain adjuncts, properly applied, looking toward the early restoration of function and the redevelopment of work habits. This last point is very important in industrial accident cases be-

cause the weekly compensation plus the desire for a lump sum settlement for permanent disability, a desire frequently abetted by legal counsel, tends to keep many cases away from work for months or years.

These adjuncts are occupational therapy, physiotherapy, personal supervision of the application of these methods by the surgeon and stimulation of the patient to try active motion and effort on his own part. Again let me repeat that most of these methods can start within a few days after the accident instead of waiting until the permanent disability is more or less fixed before starting reconstruction. Therefore, reconstructive surgery is not limited to chronic diseases and deformities.

Occupational therapy is either diversional or purposeful. It consists of work of some type given to the bed patient (bedside occupation), to the patient able to sit up and go about the hospital (hospital work—shop occupations), and to the ambulatory case (either returning to the workshop at the hospital or to workshops established away from the hospital).

Diversional occupations serve chiefly in preventing the patient dwelling on his troubles, to make him more satisfied with his hospital sojourn and to reawaken an interest in his daily contact with life.

The surliest, most grumpish patient, complaining of the carelessness of his employer for allowing such an accident, complaining of the food and surroundings at the hospital, discontent with the treatment given, can often be changed into a contented, co-operative patient by persuading him to make a scarf for his wife on one of the little bedside looms utilized for this purpose. Work furnished one patient is often contagious and soon all the men in a ward will be doing some type of work. Before Christmas several of our men patients at St. Luke's Hospital were very busy making scarfs, selling them to the nurses and doctors for from $2.00 to $5.00 each.

Purposeful occupational therapy is the prescribing of some work which will tend to restore function in a member, as the use of a plane, a hammer, a saw or a gig-saw to teach the patient to grasp with or use stiff fingers, to loosen up a stiff ankle joint, et cetera. Another type of purposeful work is that which has a vocational trend, preparing the patient to take up vocational training for a new occupation when his injury will prevent return to his old job.

The best type of occupational therapy, and the one hardest to sell to employers and to insurance companies, is to return the injured patient as soon as possible to light occupation in the plant or industry where he was formerly employed. Many an injured man, previously doing heavy, arduous work, must frequently wait months before his injury is sufficiently recovered to permit return to this heavy work. Such a one, shortly after he becomes an ambulatory case, could return to light work if his employer would co-operate and find a light job for him. It would be worth while for the insurance company to pay the wages of such a patient rather than continue to pay compensation over a period of months or even years. In the latter case this patient develops habits of idleness, becomes a pensioner, thinks constantly of a big settlement that will permit him to loaf the rest of his life or to start up in an easy business for himself and, abetted by his lawyer and family, he unconsciously endeavors to magnify his disability. Thus he becomes a traumatic neurotic case.

My experience prior to 1917, while serving as chief surgeon for a large industry, demonstrated the value of light work in the plant as the greatest form of occupational therapy and also as the best type of vocational training in many cases. Dr. Farnum, chief surgeon for the Avery Company, of Peoria, has for several years returned the majority of his fracture cases to light work within three weeks. Have you ever thought of the infrequency of traumatic neuroses among injured farmers, among business men or small store and shop keepers? These people when injured are forced to get back on the job just as soon as they become ambulatory cases.

This work, occupational therapy, is of great aid in reconstructive surgery and *must be applied early*. This idea must be sold to employers and to insurance companies if malingering and other forms of neuroses are to be reduced.

Physiotherapy, so sadly neglected by our profession prior to the war, enriching the armamentarium of all forms of quackery, has again become a recognized therapeutic agency since the war. Every hospital receiving and treating traumatic cases should have a trained physiotherapy aid to give massage, passive and active motions, hydrotherapy and function restoring exercises to such cases, under the

direction of the surgeon; and the same should be started just as early in the course of treatment as compatible with healing of the injury.

Heat is one of the best therapeutic agents. It can be applied as continuous hot fomentations, by the Burdick electric baker, by diathermy or other forms of electro-therapeutics. Elaborate apparatus is not necessary for the early treatment of injury cases. When the joint has become stiff or a member has been allowed to lose its range of motion, too often the stage reached before cases are referred for physiotherapy, then more elaborate apparatus and more prolonged daily treatments are required.

Physiotherapy misapplied, or too strenuous physiotherapy, can be detrimental to a case, increasing the permanent deformity. Again, unless carefully guarded it can slide over into the realm of quackery. Many surgeons doing chiefly industrial accident work have installed quartz lights, Burdick bakers and other forms of apparatus. Several insurance companies have put in physiotherapy units in connection with their claim departments and have employed doctors and nurses to treat chronic disabling conditions after the surgeon has completed the acute treatment. In many cases good results are obtained but too often recovery is delayed indefinitely. For example:

Recently I saw a man who had not worked for four months because of an injured shoulder. History showed that he had been carrying one end of a heavy plank on his shoulder when it slipped off and he caught it with his hand, thus giving his entire arm and shoulder a severe jerk and jar. He had been treated in the office of a doctor whose work was limited to insurance cases. Every day he went to this office and the nurse put a lamp over his shoulder and baked it for twenty minutes, followed by massage. Complaints on this date of examination were very indefinite and amounted to this: "I can't use it." Examination showed no swelling, variable points of tenderness, complete range of motion, and over the shoulder variable points of alleged anesthesia. The *x*-ray examination was negative. There was absolutely nothing wrong with the shoulder. He had developed a neurosis, which was gradually passing over into the realm of malingering.

When told that there was nothing wrong with his shoulder and that he could use it, he replied: "There must be something wrong with it or the insurance company wouldn't have paid me all these weeks and the doctor wouldn't have treated it every day." Thus the daily, easy application of a routine type of physiotherapy had allowed this case to drift into a traumatic neurosis.

Another case, recently seen, sprained his ankle, was sent to the insurance company, ankle strapped and placed under the Burdick lamp for this form of physiotherapy. He reported for daily treatments for three weeks, walking on the foot every day. Because he complained that ankle was becoming more painful, he was referred for treatments. An *x*-ray was taken before treatments were given, which showed a transverse fracture of the fibula without displacement. These examples and scores of others are convincing evidence that there must be an intelligent surgical sense directing the application of physiotherapy, otherwise it becomes a form of quackery.

Intelligent, carefully directed occupational therapy and physiotherapy after the case has become convalescent and able to be away from the hospital is equally essential for rapid recovery and restoration of function. A good physiotherapy laboratory, directed by a skilled medical man, co-operating with the surgeons who have treated the cases, is the best means of meeting the reconstructive problems during the period between hospital treatment and return to work. Because the surgeons have paid so little attention to this period of treatment, simply telling the patient to have some member of the family massage the part, a great many of these cases have drifted into the hands of the osteopaths and chiropractors. Many of these irregular practitioners have installed physiotherapy appliances and are tending to keep these methods in the field of quackery. Dr. John Coulter, working in his physiotherapy institute in Chicago and in the physiotherapy laboratories at Central Free Dispensary and Cook County Hospital, has well demonstrated that this is a scientific field belonging absolutely to the realm of medicine and surgery.

Out of the great variety of deforming conditions following trauma that have been referred for reconstructive surgery, I have chosen shoulder joint injuries to illustrate the early application of reconstructive methods with a view of preventing deformities.

Of all joint injuries referred to me for treatment, those of the shoulder joint are by far the most common except the aggravating and numerous conditions developing about the lumbar and sacro-iliac

regions. Lovett, Codman, Magnuson and Coulter and the older writers, Von Bergman, Thiem, Dittmar and others, all say in substance, "probably no other joint is so frequently injured and gives such consistent trouble as the shoulder." I would add to this that probably no other joint when injured is so consistently mistreated as the shoulder.

Von Bergman* quotes Dittmar as follows: "Of twenty-eight cases of sprain of the shoulder, only five were cured by an average treatment of 9.4 months; after 13 months in twenty-three cases there was still an average disability of 21.4 per cent." One is startled by the lengths of treatment mentioned, nine and thirteen months, for in this day of employees' compensation and of insurance companies urging rapid recovery, few surgeons persist in such long periods of treatment. Rather they tend to discharge the case before recovery is complete, assigning the lack of full restoration of function to a traumatic neurosis or malingering. One wonders, however, in reading Von Bergman's treatment of shoulder injuries if the lack of that essential factor in treatment, *the immobilization of the arm at a right angle abduction,* did not influence the prolonged period of treatment.

In shoulder joint injuries there are a few essential points in anatomy, diagnosis and treatment which every surgeon treating these cases must constantly bear in mind.

ANATOMICAL POINTS

1. There are twelve muscles passing over and around the shoulder joint and involved in its movements which can be divided into a *Strong Group* and a *Weak Group.*

The *Strong Group,* chief of which are the pectoralis major, the latisimus dorsi, subscapularis, teres major and triceps, form the strong adductors and internal rotators of the arm. The *Weak Group,* chief of which are the deltoid, the supraspinatus, infraspinatus and teres minor, form the less powerful abductors and external rotators of the arm.

2. The arm hangs vertically at the side in the position most naturally assumed by gravity. Overcoming this force of gravity becomes a most important factor when muscles long unused and atrophied, therefore weakened to a still greater extent, must again take up the burden of abducting and elevating the arm.

3. Surrounding this joint are numerous anatomical parts which may be involved in injury and interfere greatly with joint function. Thus are found numerous bursæ; many tendons, one of which, the long head of the biceps, has a sheath communicating with the joint proper; tuberosities and other bony prominences which may be torn off by direct violence or severe tendonous pull; a scapula which must be fixed for certain joint movements and must be freely movable for others; nerves and blood vessels in the axilla which frequently are involved in injury, and glands in the axilla that make this a common site for infections with resulting scars that may interfere in function.

DIAGNOSTIC POINTS

1. No joint in the body has so many parts intimately related to it as the shoulder joint. Injuries may involve several of these parts; in fact, in severe trauma it is seldom that only one of these parts is involved. Thus, a fall on the outstretched arm or a direct blow to the shoulder may cause a bursitis, a contusion of the joint surfaces and a chipping fracture of the margin of the glenoid fossa. Every part involved in the trauma must be diagnosed and considered.

2. *X*-rays of the simplest injuries of the shoulder joint are indicated. No dislocation of the shoulder should be reduced without first having an *x*-ray examination because too often a fracture of the anatomical neck of the humerus simulates perfectly a dislocation; too often a small chipped fracture of the glenoid margin complicates the dislocation or some other bone injury complicates it and means, if unrecognized, a permanently disabled shoulder joint.

If the first *x*-ray fails to show pathology and yet the symptoms persist, a second *x*-ray, two or four weeks later, often shows the calcareous deposits in a subdeltoid or subacromial bursitis, a roughening of the head of the humerus or deposits in the joint indicating a traumatic arthritis, or other slowly developing pathology. Time and again a shoulder joint case is referred to me with the statement that the *x*-ray, taken at time of injury, was negative. The case is usually called a neurosis, and when I take a second *x*-ray, the real pathological condition is very apparent, to the chagrin of the physician referring the case.

3. Failure to test the nerve sensations about the shoulder joint often accounts for failure to diagnose a nerve injury as the cause of the prolonged disability—as a circumflex nerve injury.

*System of Practical Surgery, Vol. III.

4. Persistent pain about the shoulder in the absence of all findings, frequently called neuritis, can often be accounted for by an *x*-ray of the cervical vertebræ showing an osteoarthritis as the cause.

TREATMENT POINTS

1. The first essential in treating all injuries of the shoulder joint is to protect as far as possible the weaker group of muscles, the abductors and external rotators; and to prevent contraction of the stronger group. This is accomplished by holding the arm at or a little above a right angle abduction and in partial or complete external rotation during the period of complete or partial immobilization. This can be done best by the use of a cast or by means of extension and pulley apparatus with the patient in bed, or in selected cases by some one of the airplane splints, preferably the Crane-Savenay splint.

2. Ninety per cent of the persistent disabilities of the shoulder joint are due to immobilizing the arm at the side of the body in partial or exaggerated adduction position. In young people or in certain dislocations of the shoulder joint, with only a week or ten days' immobilization before use of the arm is allowed, such a fixation may not be disastrous. But in people past middle age adduction fixation for any length of time on account of dislocation, fractured clavicle, fractured humerus or injuries in or about the shoulder joint, is absolutely contraindicated.

Try to raise the arm of an old man who has had it fixed in adduction position continuously for two weeks and note the contraction of the anterior fold (pectoralis major) and of the posterior fold (latisimus dorsi) of the axilla. Note the flabby weakened condition of the deltoid and supra spinatus. Every additional day that the arm is in adduction fixation this contraction becomes more marked and the weak abductors become less powerful.

3. If the joint has been injured so that there is danger of infection; if an arthritis or periarthritis is present; if a bursitis, which is only another form of periarthritis, exists, then the joint surfaces must be prevented from becoming adherent, or of pressing on each other, or contraction of the soft tissues or capsule about the joint must be prevented. This is accomplished by traction on the arm while the latter is held in abduction. The best means to do this is with a Thomas arm splint. A point I have recently learned from experience is to apply my traction to the upper arm, leaving the elbow joint free to move at will.

4. Early passive motion should be started in these joints as soon as compatible with healing of soft tissues or fixation in case of a fracture. Very slight passive motion can be started within the first week. Active motion must be instituted gradually and gently. Recently I saw a surgeon illustrate the use of early active motion in a fractured head of the humerus. The case had been treated excellently, but on this date, four weeks after injury, he forcibly raised the arm above its right angle fixation and rotated it. The patient complained of pain bitterly, but the surgeon insisted that early force was necessary. Never under any condition exert active or passive motion to or beyond the pain point. Early motion to the prepain point is good; beyond that you run grave risk of starting an arthritis which will undo all the benefits of a properly conducted early treatment.

5. The application of hydrotherapy in the form of large hot fomentations is always beneficial in these injured joints; likewise baking of the joint by a Burdick baker. Diathermy, or deep baking of the joint, is indicated in the later stages; early it may create too great a reaction. Massage, gentle at first and gradually increasing, but never to the painful point, can go hand in hand with passive and active motion.

The measures outlined above relate to the early treatment of shoulder joint injuries instituted with a view of preventing the permanent disabilities so commonly following these injuries. They are practically the same measures adapted to the reconstructive treatment of old disability cases referred for the purpose of restoring function in a shoulder joint. Applied early they hasten recovery without the usual prolonged period of disability necessary to regain use of the arm. Applied late, after true or false ankylosis has occurred, they require perhaps operative treatment and months of abduction treatment, physiotherapy and all the other reconstructive efforts, and even then too often fail in their purpose.

In almost every type of deformity there are reconstructive principles and methods which will improve or restore function. The early application of these methods in the surgery of trauma will prevent a vast majority of these deformities.

SOME POINTS ON THE INNERVATION OF THE CHEST*

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REFERRED PAIN

The importance of knowing the fundamental nerve supply to the chest is especially evident when one attempts to work out the mechanism involved in referred pain and the reflexes which arise from irritation of the deeper structures of the body. It may be well, then, first to review briefly the general neuro-anatomy underlying these phenomena.

General plan of a typical thoracic spinal nerve and connections with the sympathetic system.—As you recall and as again illustrated in Figure 1, a typical spinal nerve gives off three branches just peripheral to the ganglion as it leaves the intervertebral foramen or very shortly thereafter. The first of these is a small twig called the recurrent nerve, which supplies the meninges about the spinal cord. The next branch is the dorsal (posterior) primary division of the nerve, which goes to the back muscles and overlying skin. The third is a rather delicate one called the ramus communicans, but which ought to be termed the *visceral ramus*, because it carries impulses from the spinal cord to the viscera and from the viscera to the spinal cord and to the other branches of the spinal nerve. If these simple facts, as illustrated in Figure 1, were kept in mind, the horror some physicians have for the sympathetic nervous system would largely disappear. At least the great bulk, if not all, of the sympathetic ganglion cells may be regarded merely as efferent (motor and secretory) neurones which have not remained in the gray matter of the central nervous system but have migrated out nearer the structures to be innervated, necessitating nerve fibers (preganglionic) for the purpose of carrying impulses from the central nervous system to these outlying motor and secretory nerve cells (Fig. 5), just as lower centers within the central nervous system are related to descending tracts from higher centers. The clearest presentation of this subject will be found in Gaskell's[35] book on the involuntary nervous system.

*Read by invitation before the Medical Staffs of the Parkview Sanatorium and the Lymanhurst School for Tuberculous Children, November, 1923.

The only visceral afferent fibers which have been definitely identified merely course through these sympathetic ganglia and connecting plexuses without having any direct connection with the sympathetic ganglion cells and fibers coming from them. There is still some uncertainty about the terminal sympathetic plexuses; but in the main if you limit the term "sympathetic" to mean only sympathetic ganglion cells and the fibers which they produce,

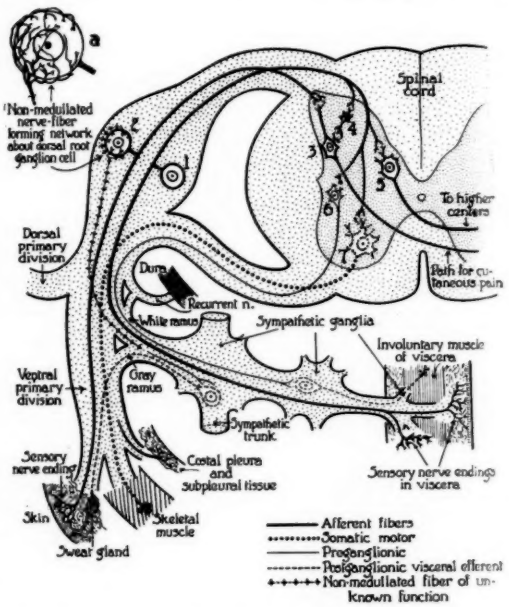

Fig. 1.—Diagram of a typical thoracic spinal nerve and connections with sympathetic ganglia: 1, visceral afferent neurone (of first order); 2, cutaneous sensory neurone (of first order, related pericellular basket shown in greater detail at a); 3, neurone of second order in the pain tract from the skin; 4, association neurone which under lowered threshold of irritability might conduct visceral sensory impulses to the cutaneous pain tract; 5, neurone of the second order in the visceral afferent path; 6, visceral efferent (motor) neurone carrying impulses out of the central nervous system to sympathetic ganglia; 7, somatic motor neurone carrying efferent impulses directly out to voluntary muscles. Further explanation in the text.

the sympathetic nervous system is motor and secretory and the two subdivisions, autonomic or parasympathetic on the one hand and sympathetic proper (Fig. 5) on the other, are terms which apply only to this efferent mechanism and not to the visceral sensory fibers that happen to follow along with them.

Each ramus communicans is usually divisible into two strands. One of these, the white ramus, is largely composed of medullated fibers consisting of large visceral afferent and finer preganglionic or visceral efferent fibers. The other bundle, the gray

ramus, is a sympathetic nerve in the strictest sense, composed almost wholly of non-medullated fibers destined for plain muscles and glands best reached by following along (and becoming mixed with) the main branches of the spinal nerves that go out to all parts of the body wall and limbs.

The main trunk of a spinal nerve (ventral or anterior primary division, shown diagrammatically in Figure 1) proceeds laterally and ventrally, supplying peripheral branches to the skin, musculature, costal pleura, etc.

From the above it is evident that afferent impulses from a particular viscus may enter a region of the spinal cord that also receives sensory impulses from a cutaneous area that is supplied by spinal nerves arising from that same region of the cord.

Theories of referred sensations.—Why a hypersensitive cutaneous area with actual pain develops as a result of irritation of some organ quite remote from the skin has been explained in various ways. The general idea is that there is a spreading of the impulses beyond the normal paths of conduction into tracts which ordinarily convey only impulses from the skin. Just where this spreading takes place is not clear.

One idea is that the non-medullated fibers which form a pericellular basket about dorsal root ganglion cells, as shown at 2 and in more detail at a in Figure 1, have been stimulated by the irritation and then transfer the impulses to a neurone whose peripheral process terminates in the skin. Central conduction would hence be the same as if the seat of the irritation were in the skin. But the fibers thus terminating as pericellular baskets in the dorsal root ganglia have never been shown to carry such impulses nor even to be visceral afferent.

A second theory is that the excessive stimulation of visceral afferent fibers lowers the threshold of irritability in the central nervous system so that impulses spread into pathways usually only involved in cutaneous stimulation. This can be readily understood by referring to Figure 1. Normally the resistance at the synapse between the visceral afferent fiber (neurone 1) and neurone 4 is so high that the path of conduction is to neurones 5 and 6; but if the resistance in the path involving association neurone 4 is greatly reduced, impulses may stray to neurone 3 and thence to the conscious center for cutaneous pain just as if the impulse originally started in the sensory fibers of the skin. Or the lowered threshold may result in impulses ascending much higher than normally but over already existing tracts. Due to lack of experience in accurately locating the seat of visceral excitation, it is falsely located in the skin connected with the particular region of the central nervous system especially closely associated neurally with the irritated viscera.

The third and least plausible explanation is that during central conduction of visceral afferent impulses, say over neurone 5 in Figure 1, the impulse spreads into an adjacent bundle of fibers, as those from neurone 3 of Figure 1, which regularly are conducting impulses from the skin. For further discussion of this subject reference may be made to Head's article listed at the end of the paper.[39]

Example of referred sensations.—The best example of referred sensations to illustrate the involved neurology is in connection with the diaphragm. As shown in Figure 2, the central portion of the diaphragm is supplied with sensory fibers by the phrenics which are branches primarily of the fourth cervical nerves. The rim and posterior third, however, is supplied by the lower six thoracic nerves. Cope[21] has definitely shown that if the phrenic region is irritated the sensation is referred to the trapezius region where the skin is supplied by the fourth cervical nerve, whereas if the periphery and posterior portion of the diaphragm is stimulated the sensation is referred to the lower chest and

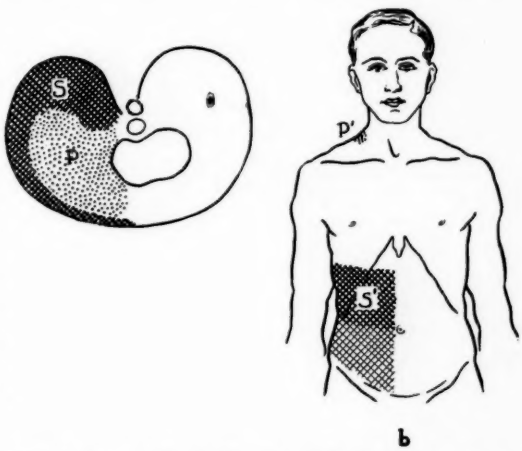

Fig. 2.—Diagram showing where pain is referred as a result of stimulating various areas on the diaphragm. a, Surface view of diaphragm; b, cutaneous areas to which diaphragmatic pain is referred. When area P is irritated the pain is located at P'. When area S is irritated the pain is referred to S'. (Modified from Capps.)

upper abdominal wall, i.e., to the cutaneous region supplied by the lower thoracic nerves. .

SUMMARY OF THE NERVE SUPPLY TO THE CHEST OF INTEREST TO CLINICIANS

The costal portion of the parietal pleura.—The costal pleura is supplied by the deeper branches of the intercostal nerves. It is sensitive to painful stimuli, but not to ordinary touch and temperature (Capps,[15] Hoffmann[41]). The pain is of the usual irritation type characteristic of peripheral nerves. It is located fairly accurately over the particular intercostal nerve involved, although localization is more accurate in the ventral and lateral regions than in the dorsal.

The diaphragm. — The phrenic nerves are the chief if not the sole motor supply to the diaphragm as is evident from the paralysis resulting from their complete severance. Blocking the phrenics with novocain has been done for relief of severe attacks of singultus (Wegele[82]), while Báron[3] has done it to stop coughing due to injury to the diaphragm. Neuhöfer[61] recommends phrenicotomy under certain other conditions where immobility of the diaphragm is desirable. A number practice it as an aid in curing pulmonary tuberculosis (Alexander,[1] Goetze,[37] Frisch[34] and others). Some indications and results of radical phrenicotomy have recently been published by Fischer.[33]

The afferent supply of the diaphragm, as has already been mentioned in connection with referred sensations, is by the phrenics and lower six thoracic spinal nerves. The reason for this dual arrangement is embryological. The central portion of the diaphragm arises from the septum transversum, which descends from the cervical region, carrying its cervical (phrenic) nerve supply with it. The periphery and dorsal portion comes from folds arising from the adjacent body wall where the lower thoracic nerves are involved.

The phrenic nerves, however, supply afferent fibers not only to both surfaces of the central and greater portion of the ventral two-thirds of the diaphragm but also to the capsule and ligaments of the liver and structures in the suprarenal region of the abdomen. Cope,[21] Orr,[63] Westphal[83] and others have recently called attention to the diagnostic value of phrenic shoulder pain in acute abdominal diseases that affect the hepatic, gastric, pancreatic and upper renal regions. Thus a point of tenderness in the neck over the right phrenic may be significant in Weil's disease (acute febrile jaundice), inflammation of the biliary tract, enlargement of the liver with stretching of the capsule, as well as diseases involving the right side of the diaphragm. Air injected into the abdominal cavity, if allowed to rise to the diaphragmatic region, produces phrenic shoulder pain.

Lungs and deep air passages.—The motor nerve fibers to the bronchial musculature arise largely from the sympathetic nerve cells of the pulmonary

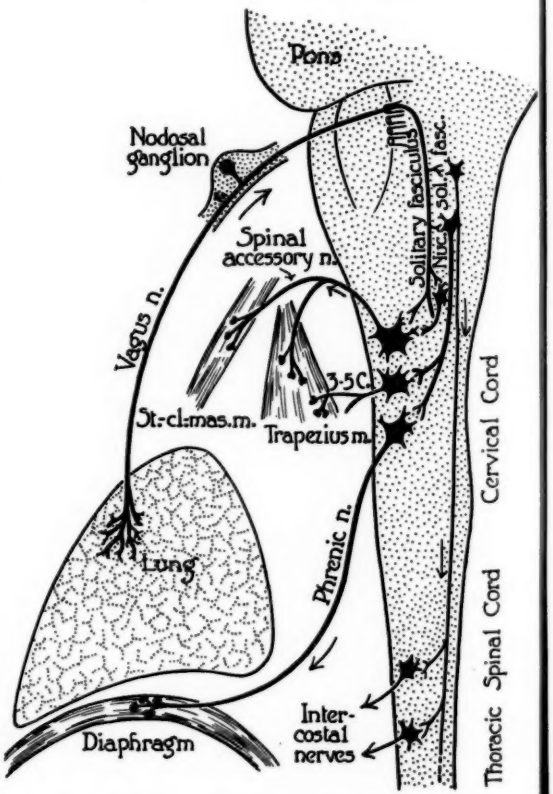

Fig. 3.—Diagram showing the probable reflex arc from the lungs over the vagus nerve to the cervical and thoracic region of the spinal cord and out to the sternocleidomastoid, trapezius, diaphragm and other muscles.

plexus outside of the lungs, but also to some extent from ganglion cells scattered along the larger bronchi, particularly as far as the origin of bronchi of the third order. These ganglion cells in turn are connected mostly with fibers from the homolateral vagus and to a slight extent with either the contralateral vagus or upper thoracic spinal nerves and sympathetic chain (Larsell,[49] Larsell and Mason[51]).

The bronchial mucous glands are apparently innervated by the same source as in case of the musculature.

The vasomotor nerves to the blood vessels of the lung are derived largely from the second and third thoracic sympathetic ganglia of the main sympathetic trunk (Fig. 5) (Möllgaard,[59] Larsell and Mason[51]). Luckhardt and Carlson,[53] however, found vasoconstrictors to the lungs in the vagus of frogs and turtles. In the turtle the vagus also contains vasodilators to the lungs. Evidently there may be variations in the vasomotor mechanism in animals of different species.

Afferent nerves and sensory endings are found in the epithelium of the primary bronchi and at the point of bifurcation of the succeeding order of bronchi nearly to the alveolar duct, in the muscles of the bronchi and in the walls of the larger pulmonary arteries (Larsell,[48, 49, 50] Dogiel[26]). The bronchoscope and even more slender instruments inserted deeply in the respiratory passages may produce coughing (Jackson,[42] Larsell[50]), although the more distal portion of the respiratory tree is less sensitive than the trachea and primary bronchi.

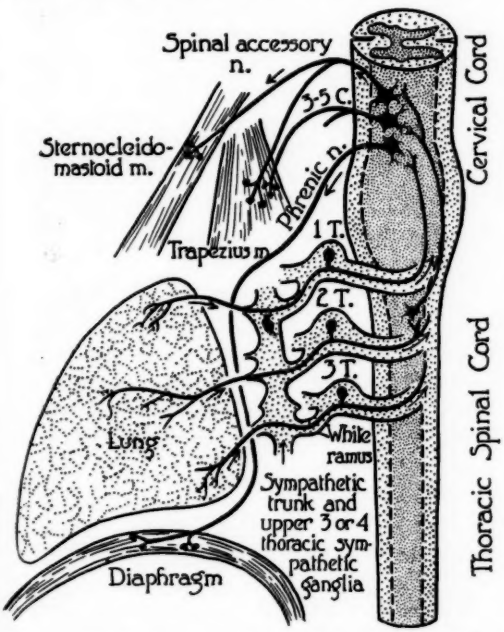

Fig. 4.—Diagram showing another probable reflex path from the lungs to the sternocleidomastoid, trapezius and diaphragm as a result of irritating the afferent fibers from the upper thoracic spinal nerves.

Most of these afferent nerves degenerate in a particular lung when the corresponding vagus is cut (Larsell and Mason,[51] Larsell[49]), indicating that they come largely from the homolateral vagus. The rest come either from the opposite vagus or from the upper thoracic nerves through the sympathetic system as shown in Figure 4. Stimulation of the deep air passages with irritating gas modifies respiration even after sections of both vagi (Craige[22]) and Pike and Coombs[65, 66] have shown that respiratory movements are markedly disturbed when the dorsal roots of the thoracic nerves are cut. These experiments emphasize the importance of these afferent fibers in the spinal nerves.

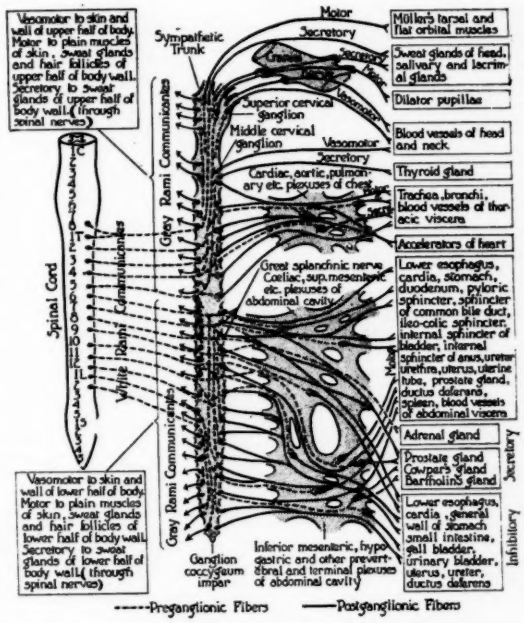

Fig. 5.—Diagram of the so-called sympathetic system proper (thoracico-lumbar outflow of general visceral efferent fibers) showing the functional significance of the sympathetic trunk and its connections.

The central connections of the afferent fibers of the vagus are diagrammed in Figure 3. This cut was designed to show the probable neurological basis for the referred pain in the cutaneous region of the fourth and adjacent cervical nerves resulting from lung disturbances and the spasticity of the trapezius, sternocleidomastoid and diaphragm in tuberculosis of the lung (Pottenger[68]). Another possibility is shown in Figure 4, although since most of the afferent fibers from the lungs (exclusive of the pulmonary pleura) are vagus in origin,

the first pathway (Fig. 3) is probably the most important.

Pulmonary pleura.—In contrast to the rest of the lung, the pulmonary pleura is supplied with afferent fibers largely, if not entirely, from the upper three or four thoracic spinal nerves (especially the second and third). These course through the white rami into the sympathetic trunk of the upper thoracic and lower cervical region, leaving the trunk in the neighborhood of the inferior cervical sympathetic ganglion (Edgeworth,[29] Möllgaard,[59] Ranson and Billingsley,[71] Hoffmann,[41] Larsell[49]). They course in the periarterial plexus along the pulmonary artery. Larsell finds that these nerves are confined to the margins of the interlobular regions and are most numerous in the dorsal margins near the hilus of the lung. They terminate in sensory endings of various types. Large areas in contact with the parietal pleura are practically devoid of nerves. This accounts for the general findings that the surface of the lung is insensitive to mechanical and chemical stimuli (Capps[15]), although Hoffmann[41] found a small area near the hilus of the lung that seemed sensitive, as one would expect from the presence of sensory nerves especially in this region. Electric stimuli may be perceived and cold (20° C.) and warmth (50° C.) cause coughing when applied to the visceral pleura. Pain is aroused only when adhesions are present, evidently because the parietal pleura becomes involved. A more careful examination of the special regions where there are sensory nerve fibers is needed.

Mediastinal or pericardial pleura.—Capps'[15] experiments on human subjects indicate that the phrenic supplies the pericardial pleura. Its irritation causes referred pain in the fourth cervical cutaneous region as in irritation of the central portion of the diaphragm and upper abdominal tissues, which are also supplied by the phrenics.

The pericardium.—This membrane is also largely supplied with afferent fibers from the phrenics, although cutting these nerves does not entirely abolish the rise in blood pressure produced by stretching or pinching the pericardium. Hence some afferent fibers evidently come from the thoracic spinal nerves via the sympathetic system (Luschka,[54] Ramstrom,[69] Capps,[15] Mathieson[56]).

Heart and great vessels.—Time will not permit any detailed statement of the motor nervous mechanism of the heart. The presence of intrinsic ganglion cells and their fibers more or less diffusely scattered as well as in groups near and within the sinoatrial node of Keith and Flack, the atrioventricular node and its continuation, the atrioventicular bundle of His, is generally recognized. These ganglion cells send fibers to the cardiac muscle cells. They receive a rich supply of pericellular nerve endings (preganglionic fibers) mostly of vagus origin.

These extrinsic nerves from the vagus are inhibitory and arise, according to Malone,[55] from the middle portion of the cell column in the medulla oblongata known as the dorsal nucleus of the vagus. Eyster and Meek[31] have obtained good evidence that the vagus normally exerts its greatest influence on the sinoatrial node, next upon the atrial portion of the atrioventricular node and still less upon the ventricular portion of the latter node. Bachmann[2] has described in considerable detail the distribution of each vagus within the heart.

The accelerator nerves leave the upper thoracic region of the spinal cord as small medullated fibers which pass through the ventral roots and white rami of the upper three or four thoracic nerves and into the sympathetic trunk, where most of them terminate about the cells of the upper thoracic and lower and middle cervical sympathetic ganglia (Fig. 5). The cardiac accelerator nerves arise from these ganglia and in connection with the cardiac fibers from the vagus form the cardiac and coronary plexuses.

A few preganglionic accelerator fibers do not end in the sympathetic ganglia of the main trunk as just described, but continue on to the cardiac ganglion of Wrisberg located in the superficial cardiac plexus. The postganglionic non-medullated fibers terminate in distinct endings on the cardiac muscle cells.

The vasomotor supply to the coronary vessels and aorta is from the upper four or five thoracic sympathetic ganglia, although there is some evidence that vagus irritation may cause spasms of the coronary arteries, thus indicating some vasoconstrictor fibers in this nerve. Langendorff (see Higier[40]), from experiments with adrenalin, concluded that the most evident effect produced by the thoracic sympathetic ganglia on the coronary vessels is dilatation.

Afferent nerves and nerve endings are abundant in the heart (Dogiel,[27] Smirnow,[79] Valedinsky,[81] Michailow[57]). Some of these fibers are from the

vagus. A special bundle to the aorta, usually designated as the depressor nerve, is of importance because the claim has been made that this can be isolated and cut with some success for the relief of angina pectoris of basal aortic origin. The rôle of the depressor nerve in slowing the heart and relaxing the arteries is well known.

Many of the afferent fibers to the heart come, however, from the upper thoracic spinal nerves, mostly over the upper three or four white rami communicantes (Ranson and Billingsley[71]). Clinical evidence indicates that they come mostly from the left side. Daniélopolu[23] found that he could relieve most of the pain in angina pectoris by cutting the dorsal root of the second thoracic nerve on the left side. In diseases of the ventricles and ascending aorta the pain is usually referred to the left upper thoracic and left third and fourth cervical cutaneous areas, although it may spread to the right side (Pottenger[68]). This is also evidence that the ventricles and base of aorta are closely connected by afferent nerves with the gray matter of the upper thoracic and mid-cervical region. Similar evidence shows that the atria are connected with the thoracic cord as low as the seventh thoracic segment. The pain in mitral stenosis with dilatation of the left atrium, for example, is referred mostly to the sixth and seventh thoracic cutaneous segments. Since the atria of the heart are derived from the venous or caudal end of the embryonal heart, the connection of the atrial portion with a more caudal region of the spinal cord is to be expected.

That these spinal fibers traverse the lower cervical and upper thoracic regions of the sympathetic trunk is supported by the relief afforded by surgical removal of these portions of sympathetic chain as has been done by Jonnesco[43] and others (Coffey and Brown,[10] Brown,[9] Brüning[11]) in angina pectoris due to aortic and cardiac lesions.

Esophagus.—The literature on the nerve supply to the esophagus has recently been exhaustively reviewed by Carlson.[17] His own experiments confirm the idea that the vagus has both motor and inhibitory control over the esophagus and cardia. If in strong tonus, vagus stimulation causes inhibition and if relaxed the same stimulus causes contraction.

Contrary to the general notion that the splanchnic nerves have nothing to do with this region of the alimentary canal, Carlson found that in the cat the same results may be obtained by splanchnic stimulation as is obtained by stimulation of the vagus. In the dog only contraction resulted, while in the rabbit only inhibitory effects were obtained from stimulating the splanchnics. Adrenalin produces both contraction and inhibition, depending upon the state of tonus at the time.

On the whole, it is necessary to assume that both the vagus and the splanchnic nerves innervate the thoracic portion of the esophagus and the cardia of the stomach. This is one more instance where the antagonism generally assumed to exist between the parasympathetic (or autonomic) and sympathetic system proper has been shown to be at least greatly overdrawn.

The sensory properties of the esophagus has been most intensively studied by Boring.[6] His results show that the mucous membrane throughout its entire length is sensitive to thermal, mechanical, chemical (alcohol and hydrochloric acid, but not to oil of peppermint nor mustard and pepper suspensions) and electrical stimuli; 60° C. gives sensations of heat, and extreme cold and heat cause pain. The upper esophagus is more sensitive than the lower portion. When the thoracic portion is excited with electrical stimuli by a small active electrode sensations are first noticeable in the arm where a large indifferent sponge electrode is applied, although this indifferent electrode covers more than fifty times as much surface as the small active electrode covers in the esophagus.

The sensations from the esophagus are generally referred too low over the lower third of the sternum and middle region of the upper half of the epigastrium when the lower third of the thoracic esophagus is stimulated. When the upper third of the thoracic esophagus is stimulated the sensations are more accurately located but usually too high and frequently in the throat region.

The afferent nerves involved are largely supplied by the vagus. The lower portion at least receives some fibers from the splanchnics (fifth to tenth thoracic spinal nerves). This explains the reference of the sensations from this region of the esophagus to the epigastrium.

The thymus.—While nothing specific can be said of the functional significance of the nerves to the thymus, the chief anatomical findings show that there are fine non-medullated nerves around the blood vessels and in the connective tissue of the capsule and trabeculæ. Sympathetic fibers from the upper thoracic and cervical sympathetic chain,

in connection with small filaments from the vagi, descend along the thyroid gland, cardiac nerves and thymic blood vessels into the gland. Occasionally some of these fibers follow the phrenic nerves for a distance (Braeucker[8]).

Apparently the thymus is insensitive to ordinary stimuli, but when inflamed it produces pain which is poorly localized. This pain may in some cases be due to encroachment upon neighboring structures such as the great vessels, lungs, phrenics, vagi, recurrent, laryngeal nerves, etc. (Phillips,[64] Lemon[52]).

The thoracic sympathetic trunk.—In connection with chest diseases, attention may finally be called to the possibility of involvement of the sympathetic trunk and related nerves, since they lie just under the parietal pleura through the greater portion of the thoracic cavity. Figure 5 gives some idea of the functional significance of this great system. It is a relay mechanism between most of the viscera of the entire body on one hand and the central nervous system on the other. The anatomical connections between the sympathetic trunk and the spinal cord are the white rami communicantes, which are limited to the thoracic and upper two or three lumbar segments, that is, almost wholly in the thoracic region. That many visceral disturbances might be brought about by the encroachment of chest abnormalities upon this system is thus apparent. Dilatation of the pupil due to pressure on the sympathetic trunk is a common example; however, this may occur from trauma to peripheral nerves in many other parts of the body (Byrne[12, 13, 14]).

REFERENCES

Since inquiries have been received from time to time for references to the literature on many of the details considered in this paper, a list of the original sources upon which this summary is largely based has been deemed worthy of publication.

1. Alexander, H.: Ueber die Bedeutung der Phrenikusausschaltung, insbesondere in Form der Exairese für die Behandlung der Lungentuberkulose. Ztschr. f. Tuberk. u. Heilstättenw. (Leipzig), 1922, xxxvi, 325-335.
2. Bachmann, G.: Distribution of the vagus nerve to the sino-auricular junction of the mammalian heart. Am. Jour. Physiol. (Baltimore), 1922, lix, 468.
3. Bâron, A.: Blockade des Nervus phrenicus bei Verletzung des Zwerchfells. Zentralbl. f. Chir. (Leipzig), 1923, l, 442-443.
4. Barry, D. T.: Afferent impressions from the respiratory mechanism. Jour. Physiol. (London), 1913, xlv, 473-481.
5. Behan, Richard J.: Pain: Its origin, conduction, perception and diagnostic significance. D. Appleton and Company, New York and London, 1921.
6. Boring, E. G.: The sensations of the alimentary canal. Am. Jour. of Psychol. (Albany, N. Y.), 1915, xxvi, 1-57.
7. Bourgery: Mémoire sur les nerfs des membranes séreuses en général, et sur ceux du péritoine en particulier chez l'homme. Compt. rend. Acad. d. sc. (Paris), 1845, xxi, 566-570, also 1380-1381.
8. Braeucker, Wilhelm: Die Nerven des Thymus. Ztsch. f. Anat. u. Entwcklngsgesch. (Berlin), 1923, lxix, 309-327.
9. Brown, P. K.: Cervical sympathectomy for angina pectoris: report of a case with dextral radiations of pain. Jour. Am. Med. Assn. (Chicago), 1923, lxxx, 1692-1693.
10. Brown, T. A.: Symptomless perforation of the esophagus. Lancet (London), 1922, cciii (vol. 2 of 1922), 1272-1273.
11. Brüning, Fritz: Die operative Behandlung der Angina pectoris durch extirpation des Halsbrustsympathicus und Bemerkungen über die operative Behandlung der abnormen Blutdrucksteigerung. Klin. Wchnschr. (Berlin), 1923, ii, 777-780.
12. Byrne, Joseph: Paradoxical pupil dilatation following lesions of the afferent paths. Am. Jour. Physiol. (Baltimore), 1921, lvi, 113-126.
13. Byrne, Joseph: Pseudo-paradoxical pupil dilatation following afferent path lesions. Am. Jour. Physiol. (Baltimore), 1922, lix, 475.
14. Byrne, Joseph: Further studies of paradoxical pupil dilatation following lesions of the afferent paths. Am. Jour. Physiol. (Baltimore), 1922, lix, 369-375.
15. Capps, Joseph A.: An experimental study of the pain sense in the pleural membranes. Arch. Int. Med. (Chicago), 1911, viii, 717-733.
16. Capps, Joseph A., and Coleman, George H.: Experimental observations on the localization of the pain sense in the parietal and diaphragmatic peritoneum. Arch. Int. Med. (Chicago), 1922, xxx, 778-789.
17. Carlson, A. J.: Studies on the visceral sensory nervous system. xiii. The innervation of the cardia and lower end of the esophagus in mammals. Am. Jour. of Physiol. (Baltimore), 1922, lxi, 14-41.
18. Carlson, A. J., and Luckhardt, A. B.: Studies on the visceral sensory nervous system. vii. Skeletal reflexes induced by stimulation of visceral afferent nerves in the frog and turtle. Am. Jour. Physiol. (Baltimore), 1921, lv, 366-384.
19. Coffey, W. B., Brown, P. K.: The surgical treatment of angina pectoris. Arch. Int. Med. (Chicago), 1923, xxxi, 200-220.
20. Cope, V. Z.: Nerve supply of parietal peritoneum and sub-peritoneal tissue. Lancet (London), 1922, ccii (vol. 1 for 1922), 415-420.
21. Cope, V. Z.: Clinical study of phrenic shoulder pain, with special bearing on diagnosis of acute abdominal disease. Brit. Jour. Surg. (Bristol), 1922, x, 192-201.
22. Craige, E. H.: The reflex produced by chemical stimulation of the deeper respiratory passages. Am. Jour. Physiol. (Baltimore), 1922, lix, 346-352.
23. Danielopolu, D.: (Resection of posterior roots in angina pectoris). Bull. Soc. med. d. hôp. (Paris), 1923, xlvii, 778-790. (Reviewed Jour. Am. Med. Assn., lxxxi, 1319.)
24. Davis, L. E.: The pathways for visceral afferent impulses within the spinal cord. Am. Jour. Physiol. (Baltimore), 1922, lix, 381-393.
25. De Witt, Lydia M.: Observations on the sino-ventricular connecting system of the mammalian heart. Anat. Rec. (Philadelphia), 1909, iii, 475-497.
26. Dogiel, A. S: Die sensiblen Nervenendigungen im Herzen und in den Blutgefässen der Säugethiere. Arch. f. mikr. Anat. (Bonn), 1898, li, 44-70.
27. Dogiel, A. S.: Zur Frage über den feineren Bau der Herzganglien des Menschen und der Säugethiere. Arch. f. mikr. Anat. (Bonn), 1899, liii, 237-281.
28. Dogiel, A. S.: Nervenendigungen in der Pleura des Menschen und der Säugethiere. Arch. f. mikr. Anat. (Bonn), 1903, lxii, 244-250.
29. Edgeworth, F. H.: On a large-fibered sensory supply of the thoracic and abdominal viscera. Jour. Physiol. (London), 1892, xiii, 261.
30. Eyster, J. A. E., and Meek, W.: The origin and conduction of the heart beat. Physiol. Rev. (Baltimore), 1921, i, 1-43.
31. Eyster, J. A. E, and Meek, W.: The permanent rhythm following destruction of the sino-auricular node. Am. Jour. Physiol. (Baltimore), 1922, lxi, 117-129.
32. Ferguson, John: The phrenic nerve. Brain (London), 1891, xiv, 282-283.
33. Fischer, H.: Indikationen und Erfolge der radikalen Phrenikotomie. Klin. Wchnschr. (Berlin), 1923, ii, 535-536.
34. Frisch, A. V.: Zur Frage der Phrenikotomie als Therapie der Lungentuberkulose. Klin. Wchnschr. (Berlin), 1923, ii, 72-73.

35. Gaskell, W. H.: The Involuntary Nervous System. London, Longmans, Green and Co., 1916.
36. Gault, C. C., and Scott, F. H.: The control of respiration. Proc. Soc. Exper. Biol. and Med. (New York City), 1922, xix, 253-254.
37. Goetze, O.: Die radikale Phrenikotomie als selbständiger therapeutischer Eingriff bei einseitiger Lungenphthise. Klin. Wchnschr. (Berlin), 1922, i, 1496-1500 and 1544-1546.
38. Head, Henry: On disturbances of sensation with especial reference to the pain of visceral disease. Brain (London), 1893, xvi, 1-133.
39. Head, Henry: Certain aspects of pain. Brit. Med. Jour. (London), Jan. 7, 1922, No. 3184, 1-5.
40. Higier, Heinrich: Vegetative Neurology. Translation by W. M. Kraus. Nervous and Mental Disease Publishing Co., New York and Washington, 1919.
41. Hoffmann, Viktor: Ueber Sensibilität innerer Organe. Mitt. a. d. Grenzgeb. d. Med. u. Chir. (Jena), 1920, xxxii, 317-328.
42. Jackson, Chevalier: Cough: Bronchoscopic observations on the cough reflex. Jour. Am. Med. Assn. (Chicago), 1922, lxxix, 1399-1403.
43. Jonnesco, T.: (Resection of the cervicothoracic sympathetics.) Bull. de l'Acad. de méd. (Paris), 1920, lxxxiv, 93, also lxxxvi, 67 and 208; Presse méd. (Paris), 1921, xxix, 193; Presse méd., 1923, xxxi, 517. His operative technique is illustrated in Presse méd., 1922, xxx, 353-357.
44. Joseph, D. R.: Experimental proof of vagus apnea. Am. Jour. Physiol. (Baltimore), 1922, lix, 491.
45. Klose, H., and Hellwig, A.: Ist die Resektion des Cervical-sympathicus eine zielbewusste Basedowoperation? Klin. Wchnschr. (Berlin), 1923, ii, 627-630.
46. Kochmann, M.: Ist das Vaguszentrum durch eine Steigerung des Blutdruckes direkt erregbar? Zentralbl. f. Physiol. (Leipzig), 1906, xx, 418-419.
47. Kohnstamm, Oscar: Zur Anatomie und Physiologie des Phrenicuskernes. Fortschr. d. Med. (Berlin), 1898, xvi, 643-653.
48. Larsell, O.: Nerve terminations in the lung of the rabbit. Jour. Comp. Neur. (Philadelphia), 1921, xxxiii, 105-131.
49. Larsell, O.: The ganglia, plexuses and nerve terminations of the mammalian lung and pleura pulmonalis. Jour. Comp. Neur. (Philadelphia), 1922, xxxv, 97-132.
50. Larsell, O.: Some aspects of the innervation of the lung. Northwest Med. (Seattle), 1922, xxii, 311-315.
51. Larsell, O., and Mason, M. I.: Experimental degeneration of the vagus nerve and its relation to the nerve terminations in the lung of the rabbit. Jour. Comp. Neur. (Philadelphia), 1921, xxxiii, 509-516.
52. Lemon, W. S.: Lesions affecting the vagus nerve. Med. Clin of North Am. (Philadelphia), 1923, vii, 293-307
53. Luckhardt, A. B., and Carlson, A. J.: Studies on the visceral sensory system. viii. On the presence of vasomotor fibers in the vagus nerve to the pulmonary vessels of the amphibian and reptilian lung. Am. Jour. Physiol. (Baltimore), 1921, lvi, 72-112.
54. Luschka, H.: Die Anatomie des Menschens (Tübingen), 1863, i, Pt. 2, 458-459.
55. Malone, E. F.: The nucleus cardiacus nervi vagi and the three distinct types of nerve cells which innervate the three different types of muscle. Am. Jour. Anat. (Philadelphia), 1913, xv, 121-127.
56. Mathieson, G. C.: The sensory fibers of the phrenic nerve. Rev. Neurol. and Psychiat. (Edinburgh), 1912, x, 553-563.
57. Michailow, Von Sergius: Die Nerven des Endocardiums. Anat. Anz. (Jena), 1908, xxxii, 87-101.
58. Molhant, M.: Les ganglions péripheriques du vague. Le Nevraxe (Louvain), 1913, xv, 525-579.
59. Möllgaard, H.: Studien über das respirationische Nervensystem bei den Wirbelthiere. Skandin. Arch. f. Physiol. (Leipzig), 1912, xxvi, 315-383.
60. Morison, Alexander: On the innervation of the sino-auricular node (Keith-Flack) and the auriculo-ventricular bundle (Kent-His). Jour. Anat. and Physiol. (London), 1912, xlvi, 319-327.
61. Neuhöfer, Paul: Ueber die Bedeutung pathologischer und kunstlicher Phrenicusschädigungen für die Einstellung und Funktion des Zwerchfells. Mitt. a. d. Grenzgeb. d. Med. u. Chir. (Jena), 1922, xxxv, 1-15.
62. Oppenheimer, B. S., and Oppenheimer, Adele: Nerve fibrils in the sino-auricular node. Jour. Exper. Med. (New York City), 1912, xvi, 613-619.
63. Orr, Thomas G.: Importance of phrenic shoulder pain in disease involving the diaphragm. Jour. Am. Med. Assn. (Chicago), 1923, lxxx, 1434-1436.
64. Phillips, John: Differential diagnosis of diseases of the mediastinum. Jour. Am. Med. Assn. (Chicago), 1922, lxxviii, 1355-1363.
65. Pike, F. H., and Coombs, H. C.: Some relations of the vagus and the spinal afferent nerves in respiratory control. Am. Jour. Physiol. (Baltimore), 1922, lix, 472.
66. Pike, F. H., and Coombs, H. C.: The organization of the nervous mechanism of respiration. Science (Garrison, N. Y.), 1922, N. S., lvi, 691-693.
67. Ponzio, F.: Le terminazioni nervose nel polmone. Anat. Anz. (Jena), 1906, xxviii, 74-80.
68. Pottenger, F. M.: Symptoms of Visceral Disease. A study of the vegetative nervous system in its relationship to clinical medicine. 2-Ed., C. V. Mosby Company, St. Louis, 1922.
69. Ramström, M.: Untersuchungen über die Nerven des Diaphragma, Anat. Hefte (Wiesbaden), Erste Abteilung, 1906, xxx, 669-695.
70. Ranson, S. W.: Afferent paths for visceral reflexes. Physiol. Rev. (Baltimore), 1921, i, 477-522.
71. Ranson, S. W., and Billingsley, P. R.: The thoracic truncus sympathicus, rami communicantes and splanchnic nerves in the cat. Jour. Comp. Neur. (Philadelphia), 1918, xxix, 405-439.
72. Schäfer, E. A.: Text-Book of Microscopic Anatomy. Quain's Elements of Anatomy, Eleventh Ed. (London), 1912, ii, Pt. 1, 325-328.
73. Shafer, E. S: Note on cats with double vagotomy. Quart. Jour. Exper. Physiol. (London), 1920, xii, 367, 1920.
74. Schafer, E. S.: The influence of the depressor nerve on the pulmonary circulation. Quart. Jour. Exper. Physiol. (London), 1920, xii, 373-394.
75. Schwartz, S.: Ueber die Lage der Ganglienzellen im Herzen der Säugethiere. Arch. f. mikr. Anat. (Bonn), 1899, liii, 63-77.
76. Scott, F. H.: On the relative parts played by nervous and chemical factors in the regulation of respiration. Jour. Physiol. (London), 1908, xxxvii, 301-326.
77. Scott, F. H., Gault, C. C., and Kennedy, R.: The regulation of respiration. Am. Jour. Physiol. (Baltimore), 1922, lix, 471-472.
78. Smirnow, A. E.: Zur Frage von der Endigung der motorischen Nerven in den Herzmuskeln der Wirbeltiere. Anat. Anz. (Jena), 1900, xviii, 105-115.
79. Smirnow, A. E.: Einige Bemerkungen über die Existenz von Ganglienzellen in den Herzventrikeln des Menschen und einiger Säugetiere. Anat. Hefte (Wiesbaden), 1904, Erste Abteilung, xxvii, 295-301.
80. Timofejew, D. A.: Ueber die Nervenendigungen im Bauchfelle und in dem Diaphragma der Saügethiere. Arch. f. mikr. Anat. (Bonn), 1902, lix, 629-646.
81. Valedinsky, I. A.: Zur Frage über die Nervenknoten im Herzventrikel einiger Säugethiere. Anat. Hefte (Wiesbaden), 1904, Erste Abteilung, xxvii, 285-293.
82. Wegele, C.: Die temporäre Ausschaltung des N. phrenicus. Deutsche med. Wchnschr. (Berlin), 1922, xlviii, 193.
83. Westphal, Karl: Muskelfunktion, Nervensystem u. Pathologie der Gallenwege. i. Untersuchungen über den Schmerzanfall der Gallenwege und seine ausstrahlenden Reflexe. Ztschr. f. klin. Med. (Berlin), 1923, xcvi, 22-51.
84. Wiggers, C. J.: The regulation of the pulmonary circulation. Physiol. Rev. (Baltimore), 1921, i, 239-268.
85. Wilson, J. Gordon: The nerves of the atrio-ventricular bundle. Anat. Rec. (Philadelphia), 1909, iii, 262-264.
86. Zak, E.: Vasomotor reflex from the aorta. Wien. Arch. f. inn. Med. (Vienna), 1922, iv, 209-234.

PRELIMINARY NOTE: A STUDY OF THE NERVOUS SYNDROME AND THE BLOOD SERUM IN PERNICIOUS ANEMIA AS AN AID IN DIAGNOSIS BEFORE RECOGNIZABLE CHANGES ARE APPARENT*

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In 1896 I reported a case of pernicious anemia[1] before the American Neurological Association at Philadelphia. The pathological findings were carefully worked out by Dr. Louis B. Wilson. The first contribution to this subject in this country was made by Putman in January, 1891. Three months later Dana reported a case; his second case was reported in 1899. My case with the neuropathological findings is the third in American literature. In 1913,[2] I read a paper before the nervous section of the A. M. A., at Minneapolis, on "Some Nervous Symptoms of Pernicious Anemia." I emphasized the great importance of these symptoms, stressing their distinctiveness, especially the subjective sensory disturbances; their constancy, their progressiveness and their early appearance. I stated that the symptoms may resemble those observed in peripheral multiple neuritis. I intimated that the sclerosis was apparently of vascular origin and might precede by months or even years the appearance of the blood picture. I illustrated this by reporting a case of Bramwell in which the cord symptoms preceded by three years the blood picture which two weeks before death showed no characteristic features of pernicious anemia. I also stated "that the personal equation determines whether the pathologic process be a pernicious anemia with or without a cord lesion or simply a diffuse degeneration of the spinal cord." "It occurs to me," says Dr. Woltmann,** "that patients may die of neurologic changes without ever developing anemia and yet have the same disease fundamentally that we refer to as pernicious anemia. The work of Hurst, Curschmann, Weinberg, and others would indicate that the achylia represents a primary constitutional familial deficiency. Naegeli has pointed out that homolysis is not always necessary and looks on a primary bone-marrow deficiency as the important conception. The laboratory evidence bears out the view expressed in your paper of 1913."

**Personal communication.

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The nervous picture in pernicious anemia is ill defined, protean, variable and progressive. Subjective sensory disturbances are usually the earliest manifestations. Beware of paresthesias, and numbness in the fingers and toes in elderly people; they may precede by months the more obvious changes. Even though the blood picture is wanting, yet the presence of such a cardinal symptom as the loss of deep sensation (loss of vibration sense, of sense of posture and inability to appreciate passive movements of fingers and toes) which is absent in only a small minority of the cases is presumptive evidence that we are dealing with this intractable malady. Touch, temperature and pain are usually normal in the early stages, not being involved until late in the course of the disease or possibly not at all. (Cadwalader.)[3] It may masquerade as a multiple peripheral neuritis. Recently I observed a case where a hysterectomy had been performed in the vain hope of curing a progressively increasing weakness and a marked sensory disturbance of two years standing. The nervous syndrome is entirely dependent upon the extent the central nervous system is involved. Taylor* states "that the cord changes are much more frequently associated than we have been accustomed to assume and this would be shown were we more skillful in our blood examinations and more accurate in our histological studies." Some time ago, I read a paper before this Association on the "Subacute Combined Degeneration of the Spinal Cord," showing that similar cord changes may occur independently of pernicious anemia due to infections and a great variety of causes. Hunter describes a case of sub-acute combined degeneration of the cord with patches of croupous enteritis in the jejunum. Some 80.6 per cent of patients suffering from this disease, according to Woltman,[4] develop nervous tissue change. Degenerative changes, once having occurred, never recede, even though there be a remission and the blood picture shows little or no abnormality; a subsequent fatal attack is only a matter of time. Courtney[5] repeatedly calls attention to the marked dissociation between the blood state and the cord lesions. He has observed the lesions go steadily on in spite of marked improvement on the part of the blood. Sensory disturbances, both subjective and objective, may occur in an apparently normal cord. (Hamilton and Nixon.)[6] Homer wrote twenty-four

*Personal communication.

books about the wanderings of Ulysses. As a college student I wished his labors had been less prolific. I do not wish to imitate him. I shall not, therefore, discuss the etiological, clinical or pathological phases of this disease. That would be Homeresque. The literature of this subject is most voluminous and is at the command of all. My purpose is to call your attention to some recent work done on the blood serum of these patients, making possible a diagnosis long before obvious changes occur, which, if confirmed, must possess a great value, not only in the early diagnosis which at present baffles the most clever clinicians, but also in enabling us to differentiate between it and the secondary anemias. In this work which has so many of the characteristics of pioneering, a healthy skepticism must be observed. No one is more conscious than my associates and myself that this preliminary note dealing with the recent work on the blood serum as an aid in early diagnosis is far from satisfactory. It will require years where we have only had months to confirm or reject this contention. The unreliableness of our present methods of procedure and the great boon it would be to both patient and doctor has seemed to us sufficient justification for our present endeavor. While it may be an illusion of hope, yet it is far from being inconceivable that if detected in its incipiency pernicious anemia might be arrested.

The original work done in this country by Putman, Dana, Wilson and others dealt with the fundamentals of its neuropathology. These have been verified and added to by subsequent workers. Recently, Dr. Hassin,[7] Chicago, has done some very remarkable histological work on multiple sclerosis and has made most interesting comparisons between the lesions of this disease and those in the cord changes of pernicious anemia. In Bramwell's case the clinical symptoms were characteristically those of multiple sclerosis. Hassin's investigations lead him to believe that pathologically the changes in subacute combined degeneration and multiple sclerosis are the same; that it is almost impossible to distinguish between them. The degenerative change in subacute combined degeneration, according to him, is "more powerful, more striking than in multiple sclerosis"; while in the former the axones degenerate in systems, in the latter they go as single fibers. (Barker.)

Globus and Strauss[8] regard the pathologic process as degenerative in character due to a toxin of unknown origin. They also state that the histologic picture is in the main similar to that found by Hassin in multiple sclerosis for this purely degenerative condition.

Taylor states that subacute combined degeneration is never truly systemic; that it is always diffuse—outside the actual system tract. Clinically one cannot differentiate between the nervous syndrome of pernicious anemia and the cord degeneration arising from other causes; if under these conditions the blood serum enables us to do this, it will be a notable advance. To the general practitioner who sees these patients first, it will be a procedure of inestimable value. Hassin suggests that future studies of multiple sclerosis must be confined to the biological, pathological and serological investigations of the spinal fluid. Because of the pathological similarities in these two diseases, a like method of research in the latter disease is worthy of consideration.

Since the degenerative process occurs in the medullary portions of the brain with about the same frequency that it does in the spinal cord,[9] mental abnormalities are common. In 650 necropsies on the insane in Michigan, Barrett found pernicious anemia present in 2.3 per cent. Cabot in 647 cases noted the incidence of mental symptoms in 15 per cent and Weisenberg states that they are present in 40 per cent of the patients (Woltman). Eighteen years ago, Langdon pointed out that the mental symptoms like those of the cord may precede the anemia. These manifestations he designated as "pre-pernicious anemia." I have seen an apparently secondary anemia overshadowed by a recurrent melancholia, only to reveal its true nature after a tonsillectomy. There is no distinctive psychosis in pernicious anemia. The mental symptoms are indicative of a "toxic-organic" process affecting the central nervous system. If the cerebral cells lack oxygen, as may occur in severe cases of pernicious anemia, permanent mental[10] changes are observed. Darden and Hall[11] think that the mental disturbances directly dependent on pernicious anemia should be classed as exhaustive or toxic psychoses. Where manic-depressive insanity and dementia precox states occur, the anemia simply acts as an exciting cause in a predisposed individual.

Constantly has the physician striven to increase his knowledge of the human body. For almost two and one-half centuries has he used the microscope to reveal its minute structure. For a century has he

employed the stethoscope to aid the ear in recognizing the sound of a disturbed mechanism. Indeed, the value of the clinical thermometer was not appreciated until the middle of the nineteenth century. The body, fundamentally, is a chemical machine. The laboratory has opened up new vistas and marvelously increased our knowledge of disease. It was the laboratory that gave us our first clear conception of trench nephritis—perhaps the greatest cause of loss of man-power during the great war—and it is through the laboratory that this study of the blood serum opens up a fascinating field of research, with an opportunity of rendering effective service to a class of long-suffering patients, with a possibility of this investigation being a milestone on the path of progress in biochemical research which ultimately will lead to a revelation of the cause of this intractable malady.

"In full fair tide let information flow—
That evil is half cured whose cause we know."

About 1920, Brockbank[12] first noticed that the color of the plasma or serum of pernicious anemia differed from that seen in the plasma or serum of normal blood or of any other anemia. Since making the observation he says, "I have used it regularly in the diagnosis of pernicious anemia and found it a most useful and reliable, if not a pathognomonic test of this anemia." The serum, according to this observer, is always a definite yellow color, varying in tint from that of a cowslip to the buttercup yellow of Canada balsam, whilst the serum of all other anemias is much paler and resembles straw-colored water." He states that from the observation of a score or more of cases of pernicious anemia, and twice the number of other anemias, a diagnosis of pernicious anemia from other forms of anemia can be made. The outstanding fact is that the serum of severe secondary anemia is a pale straw-color, not yellow. He also says that "there is no question of any pink color in the deeper colored yellow serum—nothing whatever to suggest the presence of any trace of hemoglobin or hemolysis nor is it of a bile pigment tint." The occurrence of hemolysis must be carefully guarded against. This can be avoided by "drawing 0.5 c.c. of sterile five per cent sodium citrate solution through the sterilized needle into the sterilized syringe and withdrawing the piston to its full extent, thus rinsing the barrel of the syringe with the citrate solution, which is then expelled from the syringe."

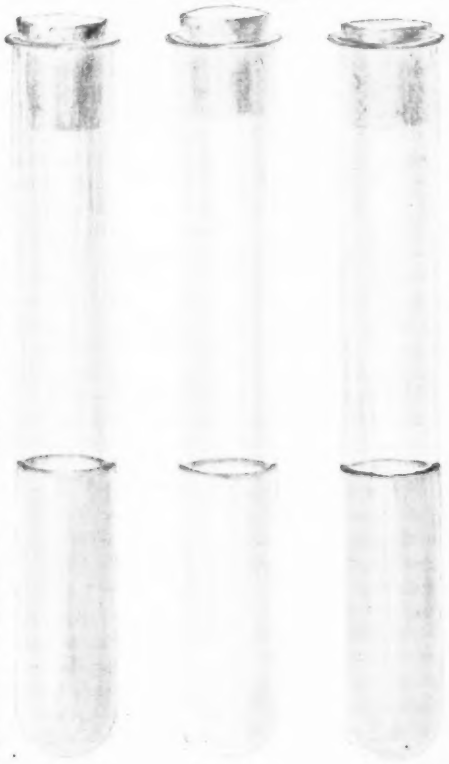
Panton, in his "Clinical Pathology" states that "the bile color of some serums is more closely simulated by the greenish yellow pigment present in the serum of nearly all cases of pernicious anemia." Stengel,[13] in a discussion of secondary hypoplastic anemia refers to the yellowish color of the blood plasma in pernicious anemia, stating that the above types present many findings of pernicious anemia, except that the blood plasma has not the definite yellow color. Brockbank thinks that the cause of this color of the serum is due to oxyhemoglobin. He demonstrated this by the examination of the serum with a pocket spectroscope which revealed two very distinct absorption bands in the position of those of oxyhemoglobin. There was a much less distinct narrow third band farther to the right in the green in some specimens. Later he checked up the pocket spectroscope with a laboratory instrument, with which the wave lengths of the definite absorption bands could be measured. In all the cases they were identical with the absorption bands of oxyhemoglobin. In normal blood serum the same absorption bands were present, though generally less distinct. No absorption bands were seen in any other form of anemia. The absorption bands were present in all specimens of pernicious anemia examined with the exception of the very pale ones. These bands are very distinct in the deeper colored serums. Continental workers have not confirmed the spectroscopic researches of Brockbank. Hematin has been observed as a pathological constituent in the serum of pernicious anemia and the yellow color has been explained by a small addition of the bilirubin to the oxyhemoglobin dissolved in the serum.

The Ariadne clue to the complete knowledge of this disease lies along the pathway of biochemistry. Much splendid work has been and still is being done along this hopeful line. Duke and Stofer[14] give as a probable explanation for the apparently relatively good color of many patients with pernicious anemia, in spite of a low blood count, the fact that the capillary red blood counts on these patients were on an average 17.6 per cent higher than similar counts on venous bloods. At the same time, the capillaries contain also an increased number of macrocytes.

Gettler and Lindeman[15] give the results of the chemical and physical analysis of the blood in 87 cases of pernicious anemia, showing such findings

as increase in non-protein nitrogen, urea and creatinin values as well as greatly increased uric acid and amino-acid content. Other interesting findings are high blood sugar, subnormal alkaline reserve, as well as low refraction and specific gravity. Similar studies, together with those of the functional capacity of the various organs, have been made by Kahn and Barsky.[16] McMaster, Rous and Larimore[17] have shown that the marked siderosis of the liver parenchyma that occurs during pernicious anemia is caused by an injurious agent derived from the gastro-intestinal tract. Recent studies by Gibson and Howard[18] show low blood and plasma cholesterol figures as well as low urea, and moderate ammonia nitrogen and high uric acid figures. A study of severe cases of secondary anemias by Mark[19] shows the similarity of the blood picture between many cases of secondary anemia and those of pernicious anemia, and the importance of developing more pathognomonic signs; while the high color index is very suggestive, recent observations have convinced him that certain patients with that disease may at sometime in their course present a low color index. Wearn, Warren and Ames[20] find that red blood cells from donors in Group IV transfused into patients in Group II with pernicious anemia and anemia secondary to nephritis remain in the circulation an average of 83 days. Ashby[21] has shown that the life of the transfused corpuscle extends thirty days or more. Denny[22] states that in ten cases of pernicious anemia the total blood volume was reduced in all but two cases, while the plasma remains normal. The examination of the duodenal contents for an increase in the urobilin and urobilinogen content in patients with pernicious anemia as emphasized by Schneider[23] and later by Giffin, Sanford and Szloppa,[24] is a valuable procedure, but this test, unfortunately, is too complicated for the use of the average practitioner. It may be regarded as confirmatory evidence not in itself diagnostic. Increased metabolic readings in pernicious anemia, as observed by a number of men, while most interesting, are disappointing as regards differential diagnosis. Leschke and Neufeld[25] find no difference in the examination of washed red blood corpuscles in pernicious anemia, secondary anemia, polycythemia, hemolytic icterus and in normal individuals. They also state that the brownish color of the blood in pernicious anemia is accordingly not referable to a degeneration of the hemoglobin, but is more probably due to the presence of waste products of the blood pigment like the dark yellow color of the serum. The responsible disintegration products of the blood pigment are methemoglobin, hematin and especially bilirubin. Dr. Bernheim* feels that the icterus index offers a very important differential point in patients with pernicious anemia, it being about 10 in the average case, whereas in secondary anemia it ranged between 2.3 and 3.9 in cases examined by her. DeWitt Stetten[26] cites several instances of pernicious anemia with an icterus index above normal.

Early Diagnosis of Pernicious Anemia from Blood Serum.



While this research work is fascinatingly interesting and possesses great potentialities and possibly the ultimate solution of this most perplexing problem, yet unfortunately it does not enable us to make a diagnosis. Happily, this is just what an observation of the blood serum does; it is a simple procedure at the command of every medical man; no annoying delay, no complicated laboratory technic. The simplicity of this method is so marked and its diagnostic importance so great, that the astounding fact is that its real significance was not recognized long ago.

*Personal communication.

The following twenty cases are excellent illustrations of the anemias, the first ten being primary, the remainder secondary. Of the former, the picture was definite and the diagnosis not to be questioned.

CASE I. J. D. Age 37 years. Past history negative. Present illness has extended over the last two years. Patient says that the first thing he noticed was shortness of breath on running up stairs. This has gradually become worse, so that now when he walks up stairs he gets extremely short of breath. If it is a long flight he must stop and rest, not from weakness but from lack of air. For the last three months patient has noticed that his strength has been getting less and that he has not been able to do his work as usual. Finally, he had to give up his position a few days ago because of his weakness. He would be extremely tired at night. On any extra exertion, for the last six months, patient has noticed that his heart would beat very fast and that he would have a dull aching precordial pain. This would last only about ten or fifteen minutes after he started to rest. Of late he has noticed that on even slight exertion this pain would develop. For the last year he has noticed numbness in legs and hands. This is worse in feet but also spreads up the legs; days that he does not have any feeling in the feet. For the last four or five weeks he has had diarrhea; having four or five watery stools at night. Was in hospital one year ago for the same trouble. After leaving the hospital he felt fine for four or five months, until about September; since then his symptoms have been getting progressively more marked. Physical examination: Marked pallor of skin and mucous membranes. Tonsils, atrophic, chronic, infected. Spleen, not palpable. Positive findings, neurological: Right knee jerk, —2 on 1, 2, 3, 4 basis; left knee jerk, —4 on 1, 2, 3, 4 basis. Achilles, right —4; left —4. Sensation, —2 —3 to pain, lower extremities on 1, 2, 3, 4 basis; —1 —2 to touch, lower extremities on 1, 2, 3, 4 basis. Vibration —4 lower extremities. Laboratory: urine negative; stools negative. Absence of free HCl in gastric contents. Blood count: Hemoglobin, 40%; r. b. c., 1,490,000; leucocytes, 4,000; differential, 100; polymorphonuclear, 56%; lymphocytes, 43%; eosinophile, 1%; poikilocytosis marked; anisocytosis marked; polychromatophilia marked; basophilic stippling marked; red cells have anemia centers. Nucleated reds present in large numbers. Diagnosis: Pernicious anemia.

CASE II. Mrs. J. D. Age 58 years. Influenza, 1918. Patient has been delicate for past eight or ten years. Under weight; has been pale as long as she can remember. Apparently not more marked recently. Moderate weakness. No history of sore tongue; no paresthesias; no numbness until one month ago, since when slight numbness of lower legs. Several attacks of upper right abdominal pain past year. Last attack one month ago and associated with vomiting. Physical examination: Marked pallor of skin and mucous membranes; malnutrition; spleen not palpable; tenderness right upper quadrant. Neurological, negative. Laboratory findings: Urine, negative; *x*-ray gallbladder shows distended gallbladder with two gallstones. Blood examination: Hemoglobin, 38%; erythrocytes, 1,740,000; leucocytes, 4,600; differential, 100; polymorphonuclear, 44; lymphocytes, 42; large mononuclear, 10; transitional, 2; eosinophile, 2; poikilocytosis, xx; anisocytosis, x. Diagnosis: 1. Pernicious anemia; 2. Cholecystitis with cholelithiasis.

CASE III. Mr. G. A. Age, 43 years. History: Present complaint, weakness and pallor. Past history negative as to illnesses or operations. Patient states that his health was very good up to about two years ago, at which time he developed a severe cold, following which he had frequent colds in his chest with indefinite body pains in his neck and arms. His present illness started about one year ago, beginning with a numbness of the legs which has been constantly present and gradually growing worse. During this time the patient has been getting weaker. About three months ago he noticed that his skin was becoming yellow. He has been taking iron ampoules and regulating his own diet but has noticed no improvement in his condition. Appetite has been good; no nausea or vomiting, no colic, bowels and stools have been normal. Cardio-respiratory: edema, palpitation and dyspnea one plus. Patient states that swelling of the feet and legs has been quite marked and that he has had to buy larger trousers and shoes because of it. Patient uses tea, coffee, tobacco and snuff. No history of sore tongue. Best weight, 170; present weight, 162. Physical examination: Tonsils buried and chronically diseased. Septum deflected moderately to the right. Teeth show alveolar abscesses. Skin shows a marked pallor of the lemon yellow type and the mucous membranes are pale. Anus shows external hemorrhoids with fissure. Poor co-operation on sensory examination (probably negative). Joint lower extremities apparently —2 —3. Vibration —3 —4. Patient's mental condition questionable. At times he seems to be somewhat irrational and at all times is uncooperative. Laboratory findings: urine, negative; blood, 6-19-23, hemoglobin, 34%; r. b. c. 1,250,000; w. b. c. 6,000; color index, 1.3, few nucleated reds, marked anisocytosis, moderate achromia, marked poikilocytosis, polychromatophilia and stippling. Diagnosis: Pernicious anemia, infected tonsils, alveolar abscess, external hemorrhoids with fissure.

CASE IV. Patient, Mr. L. W. Age 62. Present complaint is marked weakness, duration past six months. Past history is negative for illnesses and operations. For the past four years the patient has had spells of weakness followed by periods of comparative good health. Last fall, during one of the weak spells, the patient vomited and once during the winter he vomited blood. Patient states that his hands and feet have felt numb, that they feel raw now and that his legs are very weak. The patient also complains of a pain over the gallbladder region, which has been present for the last three or four years. States that the onset of the whole trouble started with a severe cold. Appetite poor with occasional nausea and vomiting. No colic, bowels constipated with stools hard. No edema, moderate palpitation and dyspnea. No history of sore tongue. Physical: Nose shows very poor drainage with a deflected septum which practically blocks the right side. Teeth, caries and pyorrhea. Tongue shows marginal smoothness. Skin sallow with a lemon yellow tint. Mucous membranes poor color. Complete neurological not done because of patient's weakened condition. Joint sense of extremities, —2 —3; vibration sense, —3. Laboratory findings: *x*-ray of stomach and heart, negative;

urine, negative; gastric analysis shows a fair digestion, mucus three plus, very slight residue, no free HCl, combined HCl 8, total acidity 8, no lactic acid, no occult blood; blood Wassermann, negative; 6-6-23, hemoglobin, 56 per cent, r. b. c. 2,200,000, marked anisocytosis, slight achromia, a few shadow forms, no nucleated reds, moderate poikilocytosis, urea 22 mg., sugar, .125 mg.; 6-13-23, hemoglobin 66 per cent, red blood cells 1,800,000; 6-25-23, hemoglobin 45 per cent, r. b. c. 1,700,000. Diagnosis: Pernicious anemia; focal infection nose and teeth.

CASE V. H. M. Age 62 years; occupation, bookkeeper. Family history, negative. Past history, hay fever every year. About March, 1921, patient noticed numbness and tingling of hands. This gradually spread to the distal third of the forearms. Patient says it felt as if he had sand between his fingers. Nine months later numbness began in the lower abdomen and then spread to both lower extremities. For the past year he has had sharp shooting pains which start in toes and fingers. These pains are severe and last about fifteen minutes. Patient has lost about thirty pounds in past six months and feels very weak. About one year ago he had a very sore tongue which persisted for three months. Has had occasional vomiting spells and appetite is poor. Does not complain of abdominal pain. Physical examination: Marked pallor of skin and mucous membranes. Tongue moderately smooth and slightly atrophic; heart, slightly enlarged to left; spleen, not palpable; liver, palpable about 3 cms. below costal margin. Neurological: positive findings; joint sense extremities —3 —4; vibration sense extremities —3 —4. Laboratory: Hemoglobin, 20 per cent; erythrocytes, 1,030,000; leucocytes, 5,600; differential, 100; polymorphonuclear, 36; lymphocytes, 44; large mononuclears, 8; transitional, 10; eosinophile, 2; poikilocytosis, double plus; anisocytosis, double plus; nucleated r. b. c. few. Wassermann, negative. Urine, negative. Diagnosis: Pernicious anemia.

CASE VI. Patient, Dr. F. S. Age 60. History: Present complaint, weakness and pallor. Past history essentially negative. Patient operated March 4, 1919, for acute intestinal obstruction. Onset of present illness two years ago, at which time the patient became tired and weak and drowsy. This feeling of weakness has been gradually increasing up to the present time. During the past month he has noticed a change of color of the skin, an increasing yellowness. He has lost five pounds in weight the past month; headache for the past month. Eyes, ears, nose and throat, negative. One year ago the patient had soreness of the mouth lasting about two weeks but there is no history of soreness of the tongue. Gastro-intestinal: Appetite has been poor; nausea but no vomiting. No jaundice; no colic; bowels have been rather constipated. Sweet things make the patient sick. Cardio-respiratory: No dyspnea; feet have been swollen past ten days. Lungs, negative. Extremities: numbness of legs past two weeks. Physical: Marked pallor of mucous membranes and lemon-yellow color of skin; heart slightly enlarged to left; edema; extremities —2 on 1, 2, 3 basis. Spleen not palpable. Neurological: knee jerks, right and left —3 —4 on 1, 2, 3, 4 basis. Achilles, —4 right and left; joint —2 —3 extremities. Vibration, —3 extremities. Laboratory findings; urine showed a slight trace of albumin; otherwise negative; blood, 6-9-23, hemoglobin, 35 per cent; r. b. c., 1,100,000; w. b. c., 6,600; marked poikilocytosis and achromia. Moderate shadow forms; anisocytosis and polychromatophilia. Blood pressure, 90/60. Diagnosis: Pernicious anemia.

CASE VII. A. D. Age 56 years. Occupation, painter. Family history: mother died of cancer of stomach. Otherwise negative. Past history: Erysipelas two years ago; hemorrhoids past ten years. No bleeding. Present history: Past eight months numbness and weakness of legs; also developed coldness which extends up to his hips. For past month these symptoms have been much worse. No soreness of tongue. Marked general weakness. Examination: marked pyorrhea; slight pallor of skin and mucous membranes. Definite baldness; slight atrophy of tongue. External and internal hemorrhoids. Neurological: Joint extremities, —3; vibration extremities, —3. Knee jerks, right, plus 1 on 1, 2, 3, 4 basis; left, plus 2 on 1, 2, 3, 4 basis. Tactile sensation, —1 to hips. Pain, —1 —2 to hips. Temperature, —1 —2 to hips. Blood: hemoglobin, 75 per cent; red blood cells, 2,370,000; color index, 1.6; leucocytes, 4,600; P. M. N.'s, 62; lymphocytes, 32; large mononuclears, 4; eosinophiles, 2; poikilocytosis, plus; anisocytosis, plus; polychromatophilia, plus. Urine, negative. Gastric analysis: total acidity, 12; free HCl, none; lactic acid, plus; occult blood, plus. Diagnosis: Pernicious anemia; subacute combined sclerosis.

CASE VIII. W. M. Age 57, laborer. Family history: Mother died of tuberculous enteritis at 28. Past history: la grippe 3 years ago; gonorrhea several times; syphilis at 17 with treatment for two months; none since. Present complaint: past ten months patient has complained of tingling in hands and legs with numbness; also progressively increasing weakness. History of sore tongue off and on. Examination: quite marked pallor of skin and mucous membranes. Definite baldness and moderate atrophy of tongue. Pupils, right —2 to light; left, —2 —3 to light on 1, 2, 3, 4 basis; liver edge 3 centimeters below right costal margin. Neurological examination: positive findings: right knee jerk, plus 2; left knee jerk, plus 2; Achilles, right, plus 2; left, plus 2; increased spasticity both legs, plus 2; both sides show Babinski, Oppenheim, Chaddock, Gordon, Mendel, Bechterew and Rossilimo; joint sense right foot, —3; left foot, —2 —3; vibration sense, right foot, —3 —4; left foot, —3 —4; moderate impairment of pain, temperature, tactile sensation both lower extremities. Laboratory examinations: urine negative except slight trace of albumin. Stool negative. Test meal total acidity 8; no free hydrochloric acid. Blood examination: hemoglobin, 24 per cent; red blood count, 1,770,000; leucocytes, 6,750; polymorphonuclear leucocytes, 78; lymphocytes, 19; large mononuclears, 2; transitionals, 1; poikilocytosis, double plus; anisocytosis, double plus. *X*-ray of stomach and colon negative. Blood Wassermann and spinal fluid negative. Diagnosis: Pernicious anemia. Subacute combined sclerosis.

CASE IX. Mrs. A. W. Age 74, widow. Past history: Negative except father and three sisters had migraine. Past diseases: typhoid at age 15; diphtheria at age 14; migraine attacks up to age 41. Present complaint: patient developed pallor eighteen months ago which has persisted. Tendency to slight sore tongue off and on during this time but lasting only a few days at a time. Has had slight creeping

sensation in hands but no definite numbness since onset of trouble. Has also had an indefinite pulling sensation of the soles of the feet but no numbness. Examination: 1. Lemon yellow tinge to skin with fairly marked pallor of mucous membranes. 2. Tongue shows areas of partial baldness. No definite atrophy. 3. Diffuse slightly elevated areas of brownish color over chest and upper abdomen and arms (Tinea versicolor). 4. Blowing systolic murmur over precordium transmitted to axilla. Heart normal in size. Neurological examination negative. Laboratory findings: Urine negative except slight trace of albumin. Blood: hemoglobin, 45 per cent; red blood count, 1,500,000; leucocytes, 3,600; polymorphonuclear leucocytes, 84; lymphocytes, 16; achromia, moderate; marked anisocytosis; marked poikilocytosis. Gastric analysis: total acidity, 6; free HCl, none. Diagnosis: 1. Pernicious anemia; 2. Mitral insufficiency; 3. Tinea versicolor.

Case X. Mr. W. S. Age 34; single; occupation, clerk. Family history: negative. Past history: typhoid at 12; tonsilitis two years ago. Present complaint: patient has developed progressively increasing pallor and weakness past four months. No history of bleeding. Has also been troubled with sore tongue much of this time. Only slight loss of weight. No gastric disturbances. Examination: Lemon yellow tinge to skin with pallor of mucous membranes; diseased tonsils; marked baldness of tongue with slight atrophy; palpable spleen; right inguinal hernia. Neurological, negative. Laboratory findings: urine negative. Blood: hemoglobin, 52 per cent; red blood count, 2,300,000; leucocytes, 4,800; differential, negative; moderate anisocytosis; moderate poikilocytosis; occasional nucleated red. Blood Wassermann, negative. Gastric analysis: total acidity, 10; free HCl, none. Diagnosis: 1, Pernicious anemia; 2, Right inguinal hernia.

Of the secondary anemias, we have unfortunately not run across one with an exceedingly low hemoglobin.

Case I. R. S. Age 33; single; occupation, laborer. Family history: negative. Past history: negative. Present complaint: past year the patient has had a persistent, indefinite dull pain slightly to the right of the navel. No radiation of pain. This pain has not been influenced by food but is somewhat worse when constipated and after exercise. No urinary symptoms. On two occasions, three months ago and two weeks ago, patient developed profuse bloody stools, bright red in color, and extending over several days. No definite abdominal pain with these spells. Following this a fairly marked pallor developed which has persisted. Examination: Marked pallor of skin and mucous membranes. Moderate tenderness over McBurney's. Urine, negative. Stools (3) negative for occult blood; *x*-ray of stomach and colon negative; proctoscopic negative. Blood hemoglobin, 40 per cent; r. b. c., 3,504,000; leucocytes, 5,700; differential, negative; moderate poikilocytosis and anisocytosis. Gastric analysis: negative in all respects. Free HCl, 22. Diagnosis: 1, Secondary anemia following hemorrhage from bowel of indeterminate origin; 2, Chronic appendicitis. At operation a chronically diseased appendix was removed. The bowel did not show any other pathology. The gallbladder was negative. The stomach showed a small duodenal ulcer.

Case II. Mrs. J. S. Age 21. Family history: Mother has migraine; one sister has always looked pale (never had blood tested, however). Past history: Appendectomy two years ago; chorea when nine years of age. Present complaint: Since three years of age patient has been subject to attacks of abdominal cramps in lower middle abdomen, coming at times as often as several times a year and again not for few years. These spells last about six hours and are associated with diarrhea, there being eight to twelve movements during each attack. No pus or blood has been observed in the stools. Was operated on two years ago, elsewhere, following a spell and the appendix was removed. No spells past six months. The patient has always been somewhat pale but especially since birth of last child. Past week patient has had twitchings of right arms and right leg and has been depressed, restless and has given expression to suicidal impulses. Also moderate weakness. No history of numbness, no paresthesias, no definite history of sore tongue. Examination: fairly marked pallor of skin and mucous membranes. Several abscessed teeth; diseased tonsils; palpable spleen; three examinations for blood and parasites were negative; *x*-rays of colon and sinuses were negative. Spinal fluid and blood Wassermann were negative. Gastric analysis was negative throughout. Hemoglobin, 57 per cent; r. b. c., 3,700,000; white blood cells, 7,400; lymphocytes, 16; transitional, 2; p. m. n., 77; polymorphonuclear eosinophiles, 1; basophiles, 1; moderate anisocytosis; marked achromophilia. The patient was on forced nourishment, tonics, and a tonsillectomy and extraction of several teeth were done. Marked improvement followed the latter two procedures, the hemoglobin being 75 per cent on discharge. Diagnosis: 1, Chorea (Sydenham's); 2, Secondary anemia (infectious in origin); 3, Diseased tonsils; 4, Abscessed teeth; 5, Acute recurring enteritis (probably infectious in origin).

Case III. C. F. S. Age 66 years. Family history: one brother died of diabetes at 70. Previous diseases: rheumatic fever fifteen years ago; several attacks. Influenza twenty-five years ago; four attacks. Present illness: Patient has always been obstinately constipated. He has also experienced a moderate degree of dyspnea for years, as well as a tendency to weakness. His health has been good otherwise up to one year ago, since when all the symptoms have been more marked. The weakness especially has been getting more progressively marked this last year, together with an increasing pallor. With the spells of constipation there is a tendency for the patient to develop vague chest pains as well as pains in the muscles of the back and a feeling of oppression over the heart. There has also been a tendency to feel depressed. There has been no history of sore tongue nor diarrhea. No numbness or paresthesias; and also no history of bleeding. For the last two years there has been a steadily increasing difficulty in walking; a block greatly exhausts him; after slight exertion feels as if the limbs would not support the body. Physical examination: the color is of a muddy pallor. No definite lemon-yellow tinge. Slight cyanosis of the lips, mucous membranes pale. Radial vessels moderately hardened but no definite calcareous deposits. Heart measure, right, 2.5 cm., left. 11.5 cm. There is a blowing systolic murmur at the apex transmitted to the axilla. There is also a soft blowing systolic

murmur at the aortic area; the aortic second sound is slightly accentuated. Spleen not palpable. Rectal examination showed moderate external and internal hemorrhoids. Blood pressure, 167-78. Laboratory: Hemoglobin, 54 per cent; r. b. c., 3,600,000; w. b. c., 6,400; moderate achromia. Marked anisocytosis; moderate poikilocytosis; moderate polychromatophilia; blood urea showed 22 mg. per 100 c.c. Blood sugar, .169 per cent. Metabolism, —5. Deep and superficial reflexes normal aside from a questionable Babinski in left foot. Vibratory sense diminished in both lower extremities, more marked in left than right. Tactile sense diminished in both feet to the ankle. Diagnosis: 1, Secondary anemia; 2, Moderate arteriosclerosis; 3, Hypertension; 4, Mitral insufficiency with slight cardiac enlargement to the left; 5, External and internal hemorrhoids.

Case IV. Miss A. B. Age 48. History of flowing off and on for last six weeks between periods. Latter have been profuse past year. Progressively increasing weakness. Examination: Fairly marked pallor of skin and mucous membranes. Uterus enlarged to size of three months' pregnancy. Hemoglobin, 37 per cent; r. b. c., 2,900,000; marked anisocytosis and poikilocytosis and moderate polychromatophilia. Diagnosis: 1, Fibroid uterus; 2, Secondary anemia.

Case V. Mr. E. E. Married. Age 55. Patient gives a history of tarry stools occurring over a period of several days in March, 1923. This recurred three weeks ago. Patient has had an indefinite distress in epigastric region for the past month not amounting to actual pain. Slight amount of gas; no vomiting. Progressively increasing weakness past month and loss of about eight to ten pounds. Examination: Pallor of skin and mucous membranes; fairly marked. Heart slightly enlarged to left; moderate external and internal hemorrhoids (no bleeding); deformity of duodenal bulb; no retention in stomach. Hemoglobin, 46 per cent; r. b. c., 3,000,000; w. b. c., 7,800; marked anisocytosis and poikilocytosis. Free HCl 3. Gastric analysis otherwise negative. A diagnosis of probable duodenal ulcer and secondary anemia was made and the patient explored. At operation the pancreas was found to be about three times its natural size, fairly hard and impinging on the duodenum, causing the above deformity. The gallbladder, which was thick-walled and bound down by adhesions, as well as a moderately diseased appendix, was removed. It was the opinion of the surgeon that the pancreatic condition was that of a chronic pancreatitis. Subsequent examination—about two months later—showed the patient much improved with a hemoglobin of 75 per cent and red blood count of 4,400,000.

Case VI. Mr. J. S. Age 54. Occupation, miner. Married. Family history: Mother died at 55, heart disease. Past history: pneumonia. Present history: past eight months epigastric distress daily, coming on right after eating. Previous to this had had epigastric distress off and on one to two hours after meals with food relief. With distress now has gas and bloating. No vomiting. Loss of fifteen pounds in weight with increasing weakness and pallor. Examination: Moderate pallor of skin and mucous membranes. Mass in epigastric region; diseased tonsils; one abscessed tooth. Laboratory findings: urine negative. Gastric analysis: total acidity, 67; free HCl, none. Wassermann, negative. Blood: hemoglobin, 56 per cent; r. b. c., 2,400,000; leucocytes, 5,600; differential, negative. *X*-ray of stomach showed marked filling defect of pyloric end of stomach. Diagnosis: 1, Cancer of stomach (inoperable); 2, Secondary anemia.

Case VII. W. P. Age 65 years. Family history, negative. Past history: Rheumatic fever every year past four years. Present complaint: past three weeks patient has had generalized swelling of joints. Practically all large joints moderately swollen, with stiffness, slight redness and considerable pain. Temperature up to 102. No sore throat. Patient has been rather pale the past year. No history of bleeding. Examination: Moderate swelling of right knee and left wrist with limitation of movement and slight redness. Moderate pallor of skin and mucous membranes. Laboratory findings: Urine negative. Blood: hemoglobin, 55 per cent; r. b. c., 3,050,000; w. b. c., 13,000; p. m. n., 49 per cent; lymphocytes, 48 per cent; eosinophiles, 1; slight poikilocytosis and anisocytosis; basophiles, 2. Diagnosis: 1, Acute polyarthritis; 2, Secondary anemia.

Case VIII. Mrs. G. L. Age 34. Married. This patient gives a history of impaired health the past two years, manifesting itself chiefly as weakness and anorexia. During this time she has had spells of indefinite fever, the temperature going up to 103 towards the end of the day for a period of several weeks. At these times the weakness is much more pronounced and she is confined to bed. With one of these spells there was an associated attack of abdominal pain and her appendix was removed elsewhere. There is no history of diarrhea; no cough, etc. On examination the following findings were found: marked pallor of skin and mucous membranes. Nine abscessed teeth. Diseased tonsils; moderate abdominal distension and generalized tenderness. Blood count: Hemoglobin, 58 per cent; r. b. c., 3,460,000; w. b. c., 4,400; lymphocytes, 38 per cent; large mononuclears, 3 per cent; p. m. n., 59 per cent. Moderate anisocytosis and poikilocytosis. Examinations such as blood Wassermann, *x*-ray of chest, investigation for plasmodia, daily four-hour temperature and pulse rate over a week, etc., were negative. Diagnosis: 1, Secondary anemia of septic origin; 2, Alveolar abscesses; 3, Diseased tonsils. The patient was put to bed; extraction of teeth and tonsillectomy were done. She gradually improved on tonics and forced nourishment and when discharged the hemoglobin was 76 per cent.

Case IX. Mrs. J. S. Age 34; married. Occupation, housewife. Family history, negative. Past history: pneumonia four years ago; influenza two years ago. Present complaint: bloody stools off and on past few years. Up to four months ago, only occasional spells when bright red blood in stools. Past four months small amount of blood in stools practically daily. General health fairly good except somewhat weak. Examination: Moderate pallor of skin and mucous membranes. External and internal hemorrhoids. Laboratory findings: Blood: Hemoglobin, 46 per cent; r. b. c., 3,200,000; differential count negative; slight anisocytosis and poikilocytosis. Urine, negative. Diagnosis: 1, Internal and external hemorrhoids, bleeding type; 2, Secondary anemia due to bleeding hemorrhoids.

Case X. Mr. J. C. Age 64; married. Occupation, laborer. Family history: Mother died at 50 of heart trouble; father died at 60 of nephritis. Past illnesses:

none. Present complaint: Past nine months patient has been getting progressively weaker and losing weight—loss of 50 lbs. Developed jaundice at onset of trouble, which has become gradually more marked. Noticed a mass in upper right abdomen which has increased in size. Has had progressively increasing gastric distress as dull pain in epigastric region immediately after eating, with occasional eructations of food. Examination: Marked generalized jaundice; marked pyorrhea; emaciation; pallor of mucous membranes; liver extending 8 cm. below right costal margin, hard and irregular. Laboratory findings: Gastric *x*-ray shows filling defect at pyloric end of stomach. Blood Wassermann, negative. Urine negative except for trace of albumin. Blood: Hemoglobin, 50 per cent; r. b. c., 4,120,000; leucocytes, 9,400; differential, negative; slight anisocytosis and poikilocytosis. Gastric analysis: total acidity, 8; free HCl, none; blood, positive; Boas-Oppler bacilli present. Diagnosis: Cancer of stomach with metastasis of liver; obstructive jaundice; secondary anemia.

The above cases are sufficiently basic and are certainly representative of the anemias, while at the same time they happily exemplify the specific conditions as to the color of the blood serum and its differential significance. The contrast in color is so marked that one cannot mistake it. The yellow color of the blood serum in pernicious anemia may even show a slight greenish tinge with the yellow. Brockbank states there has been no greenish tint in any of his cases. The straw color of secondary anemia varies from a pale straw to a deep straw, depending upon the degree of anemia present; the color of the serum of normal blood is a pale yellow of deeper tinge than that of secondary anemia but not as deep a yellow as that of pernicious. Occasionally a normal serum may approach the color of pernicious anemia, but after having observed several specimens of the latter, the differentiation is not difficult. Spectroscopically, the serum of primary anemia absorbs almost completely the violet end of the spectrum but Dr. Kvitrud has not been able to demonstrate distinct bands of oxyhemoglobin between the D and E lines of the spectrum, and Prof. Joseph Valasek of the Department of Physics, University of Minnesota, has confirmed his findings. Normal serum absorbs a little of the violet at the extreme end but never as much as pernicious anemia. If the serum of the latter is kept in a sealed tube in a few days it begins to lose its color. Unsealed, change is apparent in 24 hours.

Ten years ago I emphasized the importance of the recognition of the nervous syndrome in pernicious anemia, as an aid in diagnosis. Today I am calling attention to a study of the blood serum in this disease as a positive and more reliable aid in diagnosis even before recognizable changes become apparent. It also enables one to differentiate the nervous disturbances of primary anemia from those due to other toxic influences. There is a feeling that an intractable disease of this character is hopeless, that palliative measures only are indicated, that a possibility of a discovery of its cause and using appropriate therapy is merely a pious hope—the dream of a visionary. It is well to recall what Sir Francis Bacon says regarding supposedly incurable disease—"A work therefore is wanting upon the cures of reported incurable disease that physicians of eminence and resolution may be encouraged and excited to pursue this matter, as far as the nature of things will permit, since to pronounce incurable is to establish negligence and carelessness, as it were, by law and screen ignorance from reproach."

Biochemistry* has greatly enriched our knowledge of this disease; it has not, however, measurably increased our diagnostic acumen. Humiliating though it be, yet we do not know any more about the etiology and cure of this disease than we did one hundred and one years ago when Combe reported the first case. Those were immortal words with which John Hunter addressed Jenner when he was thinking of investigating vaccination—"Do not think, but try; be patient, be accurate."

To the laboratory, nevertheless, we are to look for the ultimate revelation of the pathogenic factor and a subsequent rational therapy. We must not forget, however, that attractive theories too frequently are like glow-worms; they seem brighter from afar. We should never lose sight of the fact "that the history of progress is the attainment of the supposedly impossible." "Before a discovery can be made," says DaCosta,[27] "the scientific imagination of some one must flash into Auroral hues."

CONCLUSIONS

1. The nervous disturbances in pernicious anemia are characteristic and distinctive and are observed in over 80 per cent of the cases. Their presence should always suggest the probable occurrence of this disease.

2. The yellow or greenish yellow color of the blood serum in pernicious anemia is easily recog-

*The blood test of Dr. Shaw-MacKenzie, by means of which he has been able to differentiate cancer of internal organs from other non-cancerous conditions in the majority of cases and in obscure cases to exclude the presence of cancer, is believed to possess great diagnostic possibilities. In a total of 221 serums examined, the method has proved correct in 211 or 95.5 per cent. (Simpson.)

nized by the naked eye and is a positive—one might say with Brockbank — *almost a pathognomonic symptom* of this disease.

3. The blood serum of secondary anemia varies from a pale to a deep straw color and, when contrasted with that of the pernicious type, a failure to distinguish between them is not possible.

4. The color of normal blood serum is a pale yellow, has a deeper tinge than that of the secondary form and while it may occasionally resemble it yet after a little experience differentiation is simple.

5. The absorption bands of oxyhemoglobin, described by Brockbank, as explaining the yellow color of the serum in primary anemia, Dr. Kvitrud has been unable spectroscopically to demonstrate; his observations have been confirmed by Prof. Joseph Valasek, Department of Physics, University of Minnesota.

6. The buttercup yellow in the blood serum of primary anemia is readily seen by the unaided eye. With a little experience the practitioner can, by its observation, make a diagnosis of this disease before definite changes manifest themselves.

7. The blood serum in the border-line cases requires patient research. A later contribution may be expected.

NOTE: I am greatly indebted to Dr. Gilbert Kvitrud, Director of the Laboratory of Mounds Park Sanitarium, for painstaking research, and to Dr. Arthur E. Mark, for his aid in the collection of cases and review of the literature. Without their assistance this paper would not have been written. To Miss Hirsch of the Department of Anatomy, I am indebted for the drawings of the blood serum from the actual specimens, and to my friend, Dr. Frank E. Burch, for the excellent colored slides.

BIBLIOGRAPHY

1. Riggs, C. Eugene: The spinal cord in a case of pernicious anemia. Int. Med. Mag., Sept., 1896.
2. Riggs, C. Eugene: Some nervous symptoms of pernicious anemia. Jour. Am. Med. Assn., Aug. 16, 1913.
3. Cadwalader, W. B.: On the diagnosis of sub-acute combined sclerosis of the spinal cord associated with severe anemia. Jour. Nerv. & Mental Disease, 1916.
4. Woltman, H. W.: Nervous symptoms in pernicious anemia. Amer. Jour. Med. Sc., 1919.
5. Courtney, J. W.: Boston Society of Psychiatry and Neurology. Discussion of R. M. Shukles' paper on "Nervous Symptoms of Pernicious Anemia." Jour. Nerv. & Mental Disease, October, 1922.
6. Hamilton, A. S., and Nixon, C. E.: Sensory changes in sub-acute combined degeneration of pernicious anemia. Arch. of Neurol. and Psychiat., July, 1921.
7. Hassin, G. B.: Pathological studies in the pathogenesis of multiple sclerosis. Association for Research in Nervous and Mental Dis., vol. ii, 1921.
8. Globus, J. H., and Strauss, Israel: Progressive funicular myelopathy (Sub-acute combined degeneration). Arch. Neur. & Psych., October, 1922.
9. Woltman, H. W.: Brain changes associated with pernicious anemia. Arch. Int. Med., June, 1918.
10. Editorial, Jour. Am. Med. Assn., June 23, 1923.
11. Darden, O. B., and Hall, J. K.: The psychoses associated with pernicious anemia. South. Med. and Surg., November, 1922.
12. Brockbank, E. M.: The blood serum in pernicious anemia. Brit. Med. Jour., July 22, 1922.
13. Stengel, A.: Types of severe anemia. Jour. Iowa State Med. Soc., December, 1922.
14. Duke, W. W., and Stofer, D. D.: A comparison of capillary and venous blood in pernicious anemia. Arch. of Int. Med., Vol. 30, No. 1, July, 1922.
15. Gettler, A. O., and Lindeman, E.: Blood chemistry of pernicious anemia. Arch. Int. Med., Vol. 26, Oct. 15, 1920.
16. Kahn, M., and Barsky, J.: Studies of the chemistry of pernicious anemia. Arch. Int. Med., Vol. 23, 334, March, 1919.
17. McMaster, P. D., Rous, R., and Larimore, L. C.: Significance of hemisiderosis of pernicious anemia. Jour. Exper. Med., Vol. 35, 1922. (Date not given.)
18. Gibson, R. B., and Howard, C. P.: Metabolic studies in pernicious anemia. Arch. Int. Med., Vol. 32, July, 1923, pp. 1-16.
19. Mark, A. E.: A study of severe secondary anemia. Minn. Med., Sept., 1922.
20. Wearn, J. T., Warren, S., and Ames, O.: Length of life of transfused erythrocytes in patient with primary and secondary anemia. Arch. Int. Med., 29, 527, 1922.
21. Ashby, Winifred: Determination of the life of transfused blood corpuscles in Man. Jour. Exper. Med., Vol. 29, 1919. Jour. Exper. Med., Vol. 34, 1921.
22. Denny, G. P.: Blood volume in pernicious anemia. Arch. Int. Med., Vol. 27, 1921.
23. Schneider, J. P.: The splenic pathology of pernicious anemia and allied conditions. Arch. Int. Med., Vol. 17, 1916.
24. Giffin, H. Z., Sanford, H. H., and Szloppa, X.: Estimation of urobilin and urobilinogen in the duodenal contents. Am. Jour. Med. Sc., 1918.
25. Leschke, L., and Neufeld, V.: Untersuchungen über den Blutfarbstoff beim gesunden und blutkranken Menschen. Klin. therap. Wchnschr 1; 849; 1922; Ztschr. f. klin. med. 94, 224, 1922.
26. Stetten, DeWitt: The surgical value of the estimation of the bile pigmentation (icterus index) of the blood serum. Ann. Surg., Aug., 1922, Vol. lxxvi, pp. 191-200.
27. DaCosta, C. J.: Behind the office doors. Ohio State Med. Jour, July 15, 1911.

DISCUSSION

DR. GILBERT KVITRUD, St. Paul: Dr. Brockbank says that he finds the absorption bands of oxyhemoglobin in the serums of most of his pernicious anemia patients. I have examined the serums of ten or more primary anemias and have as yet not been able to find distinct bands between

the D and E lines of the spectrum when using an absorption cell 1 cm. thick. The only absorption seen with the two prism indirect spectroscope is at the violet end of the spectrum, the violet being usually completely absorbed.

Drs. Riggs, Mark and myself have studied the serums from a number of pernicious and secondary anemia patients as to color and agree with Dr. Brockbank that these serums have a characteristic pigment. The color of a secondary anemia serum is pale straw, while that of primary anemia is deep yellow and at times a greenish yellow. We do not know what this pigment is or why it should appear in serums of pernicious anemia.

We will have something to say of the border line cases at a future date.

DR. CHARLES R. BALL, St. Paul: Mr. Chairman, Ladies and Gentlemen: I think we should be very grateful to Dr. Riggs for calling our attention to another diagnostic point in the early recognition of pernicious anemia. I confess that in most of the cases which come under my observation a diagnosis from the blood elements alone at the time I see them is very difficult. The blood picture may be suspicious but lacks very much of being pathognomonic.

For many years I have depended, in my recognition of this disease, more on the nervous symptoms than I have on the blood findings. In those cases which manifest nervous symptoms, these seem to be quite pathognomonic of this condition.

Chief among these symptoms is a numbness in the fingers, and on the soles of the feet; the patients complain very much of this numbness and in the use of their hands and fingers are very clumsy. In the female patients, they complain that they are unable longer to sew because they cannot hold a needle.

In the men, they say they are unable to button their clothes and if you ask them to attempt to do this, you see that they are fumbling constantly for the button and the button-holes. The sensation in their finger tips is not sufficient for them to recognize these distinctions.

Also, there is an exquisite sensitiveness, quite general all over the whole body but perhaps best exemplified in the calves of the legs. You can bring this out nicely by pinching the calves.

Then, there is quite a marked disturbance in balance.

In the connection in which these symptoms are found, it is not difficult to differentiate pernicious amenia findings from other disease. I cannot think of any condition which presents quite this syndrome in the background in which it occurs.

Then, the tongue has been quite an important factor in the aid to diagnosis. You all know the characteristic tongue so often seen in pernicious anemia, the rather small atrophic looking tongue which is red and perfectly clean and sometimes sore around the edges. This is a factor which should never be neglected in making a diagnosis.

I used to think that the nervous symptoms were due to the anemic condition in the blood. I have long ago given up this idea.

In my opinion, the nervous findings are due to the same toxic agent which causes the blood changes. Sometimes this toxic agent depresses the blood almost entirely and does very little damage to the nervous elements, while in other cases the situation is exactly reversed. The nervous elements are the ones which seem to be affected the most and the blood changes are moderate or perhaps none which one is able to recognize.

Two years ago I had two very interesting cases occurring in a man and his wife. The man had a high grade of anemia and very few nervous symptoms. The wife had no blood changes whatever but marked nervous disturbances such as one would expect in a case presenting a combined sclerosis.

These two cases of pernicious anemia occurring in man and wife makes one wonder if there may not be some tendency of an infectious nature in this condition.

If this serum test, which Dr. Riggs has presented to us, proves to be reliable, it will make the early recognition of cases of pernicious amenia much easier than it is at present.

DR. HENRY WOLTMAN, Rochester: It is given to very few men to see their contributions withstand the weathering influences of 27 years. Dr. Riggs was the first in this country to report a case of pernicious anemia with microscopic examination of the central nervous system and we find that his observations stand today as they did then. Prophecy is a dangerous business for the prophet, but here, too, he has succeeded for his assertion that the examination of the nervous system would prove to be a valuable aid in the early diagnosis of pernicious anemia has been amply verified.

Recent evidence has been adduced by Martius, Weinberg, Hurst and others, that goes to show that the achlorhydria, the *sine qua non* of pernicious anemia, is not the result of of an atrophy of the gastric mucous membrane, but represents a primary, constitutional, familial deficiency, present from infancy. It is only in these cases, supposedly, that pernicious anemia develops. Grawitz, furthermore, has demonstrated that hemolysis is often lacking, and regards a primary bone marrow deficiency as the important conception. This conception, too, is in line with the suggestion first made by Dr. Riggs, that individual predisposition probably determines which patients are going to develop degenerative changes in their nervous systems.

The clinical changes defied all explanation on the basis of known cord involvement alone, and it was not until Hamilton and Nixon demonstrated the frequency of multiple peripheral nerve degenerations that the problem reached solution. This fine piece of work, and Barrett's demonstration of Lichtheim foci in the brain, are the two outstanding contributions to our knowledge of the pathology of pernicious anemia since Lichtheim's original work.

I am not qualified to discuss the color reactions of the serum as described by Dr. Riggs, save to say that because of its simplicity it should prove to be of the greatest practical value to the general practitioner and hence of the greatest benefit to the patient.

Some years ago Blankenhorn called attention to the marked increase in bile pigments in the serum of pernicious anemia patients and stated that the intensity of the reaction ran parallel to the neurotoxicity exhibited by the case. I should like to ask Dr. Riggs whether the intensity of the color of the serum shows any parallelism to the neurotoxicity?

In 1904, Syllaba called attention to pre-pernicious anemia icterus. Is there any parallelism between the color of the serum and the intensity of the icterus?

There is always a tendency to attribute too much

specificity to a new test. Dr. Riggs warns against this, but may I ask whether this reaction has been observed in any other conditions, such as congenital hemolytic icterus? Possibly not enough cases have as yet been observed to answer this question.

Finally, may I ask about the color of the spinal fluid when this reaction is present?

DR. H. M. CONNER, Rochester: I also should like to ask Dr. Riggs if this color change is any different in pernicious anemia from that which occurs in any of the hemolytic anemias, such as, for instance, in hemolytic jaundice which Dr. Woltman has mentioned; also whether the color can be distinguished from that which occurs in any case of jaundice? Last spring it was my pleasure to talk with Dr. Bernheim, whom Dr. Riggs mentioned, regarding the color changes in the serum. At that time Dr. Bernheim was not able to distinguish between the color changes in jaundice and those occurring in hemolytic anemias. On the face of things, knowing that in the hemolytic anemias we have increased destruction of the red blood cells, it would appear that this color change is due either to the liberated hemoglobin or to the bile pigments formed from the excess hemoglobin. If this is true, one would not be able to make this test a diagnostic feature of pernicious anemia.

DR. C. EUGENE RIGGS (closing): When I was a boy in Sunday School I remember we had a lesson in which angels were spoken of. I asked the teacher, a very distinguished clergyman, if he knew what an angel looked like. He smiled and he said: "I don't, do you?" You understand this is a preliminary note, and these things that my good friend Woltman and the speaker following him emphasized, we have not had time to investigate. We have only had time to go into the simple facts that we have given to you today, and you will remember that I said that other communications might be expected.

I am sorry, but I will have to give you the same answer that the Sunday School teacher gave me, Dr. Woltman, with reference to the questions you have asked. But we feel that if there is anything in this contention of Brockbank it is an especial boon to the general practitioner. It is something that as medical men we ought to investigate and consider with a critical judgment and reject if we find that our hopes have not been realized.

The last speaker asked with reference to the vibration test. That, of course, we very definitely find in primary anemia. I have forgotten the other question.

THE CHAIRMAN: Sense of posture.

DR. RIGGS: In this little abstract I gave you I could not make any reference to this because of lack of time. I might say that the loss of deep sensibility is the most important differential point from a nervous standpoint. Dr. Kvitrud will explain to you the preparation of this serum. If you get hematolysis it vitiates the diagnosis. I would like to say that these drawings which the doctor has are very much more satisfactory in every way than our slides and they were made by the artist at the University in the Department of Anatomy and taken from the actual laboratory specimens.

DR. GILBERT KVITRUD: Of course, this blood has to be obtained in such a way that there is no hematolysis and in order to do this we use a sterile syringe and rinse it out with a five per cent sterile sodium citrate solution. We obtain 5 c.c. of the blood and inject it into a test tube carefully so that there will be no mechanical hematolysis. Then put the tube in a slanting position so that the serum can separate out better than if the tube was upright. We usually leave it over night and then draw off the serum the next day and centrifuge it for quite a while with a high speed centrifuge. After that, it is ready for the examination by the spectroscope or grossly as to color.

It would be pretty hard to transport the blood. If you went some place to get it you would almost have to leave it there until the serum separated out, and then you could take the serum and centrifuge it when you get to your hospital or office.

SILICA IN TUBERCULOSIS

Compounds of silica have found a place during recent years among drugs of reputed value in the treatment of tuberculosis. In view of their extreme insolubility, one would scarcely expect them to exert any immediate pharmacodynamic effect. Nevertheless, there is evidence that silica finds its way into the tissues and organs and remains deposited, notably in connective tissues. This has given rise to the hypothesis that the element plays a part in determining the elasticity and tensile strength of fibrous tissues, although the smallness of the quantities of silica ordinarily found should make one extremely skeptical of the validity of any conclusion of this sort. Nevertheless, various silica-containing teas or drugs have been recommended in the hope that they would increase the amount or improve the quality of the connective tissue that forms the defense about tuberculous lesions. A study at the Sprague Institute in Chicago by Maver and Wells brought nothing but negative results through the administration of silica preparations to tuberculous animals.—*Jour. A. M. A., May 17, 1924, p. 1610.*

PROMONTA NOT ACCEPTED FOR N. N. R.

Promonta (manufactured by the Chemische Fabrik Promonta G.m.b.H., Hamburg, Germany, and distributed in the United States by the Acme Pharmaceutical Co., Chicago) is said to be "an organic preparation from nervous matter of the central nervous system combined with polyvalent vitamines, lime, iron, hemoglobin and soluble albuminoids." Promonta is reminiscent of the "Nerve, Brain and Skin foods," "nerve tonics," etc., which had their vogue in the United States before the passage of the Food and Drugs Act; like them, it is recommended for "neurasthenia," "all kinds of fatigue and exhaustion," "anemia," "bloodlessness," "impaired vitality," etc. The inclusion of "4 per cent" of "polyvalent vitamines" (source not stated) is a modern touch, as is also the reference to experiments on animals (with pictures) which had been made to grow more rapidly by the addition of "Promonta" to their usual diet. Promonta is an irrational mixture of secret composition exploited under preposterous claims.—*Jour. A. M. A., May 24, 1924, p. 1712.*

OBSERVATIONS ON SOME OF THE MORE RECENT PROBLEMS IN RHINOLOGY*

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The history of medicine gives no more interesting record than that of the various troubles attributed at one time or another to nasal conditions. The rather complicated structure of the nasal cavities, especially those of the ethmoid, and the relation of these cavities and accessory sinuses to important structures such as the orbit, nerve trunks and the brain cavity, all combine to render plausible, or better perhaps, possible, some more or less satisfactory anatomical basis for explaining symptoms attributed to the nose.

The discussion over nasal reflexes which began in 1871 when Voltolini reported two cases of typical bronchial asthma cured by removal of nasal polypi, resulted ten years later in a flood of contributions to the literature discussing the subject of reflex nasal neuroses in which were expressed the wildest prophecies regarding the extent of the nasal relation to other diseases. There were those who seemed ready to believe that there was scarcely any ailment that might not be cured by treating the nose. Many rhinologists of those days accepted the view expressed by Bosworth that all cases of spasmodic asthma are associated with pathological conditions in the nasal cavities and, furthermore, that the source of asthma can always be traced to these nasal conditions. It is quite easy for all of us today to agree with the first of these propositions, namely, that all cases of asthma are associated with palpable evidence of pathological changes in the nasal cavities or sinuses, particularly if we are permitted to include such conditions as the usual irregularities of the septum and the ordinary turgescence of the turbinated bodies. The genuine enthusiast on nasal neuroses did not even stop at palpable evidence of abnormal conditions in the nose, but advocated the existence of hypersensitive spots in the mucous membrane of the septum and turbinated bodies in noses, that appeared on inspection quite normal, from which these supposed nasal reflexes might arise.

What was claimed in the eighties regarding the relation between asthma and the existence of palpable changes in the structure of the nasal cavities, we can all accept regarding such conditions as headache, eye symptoms, various so-called neuralgic syndromes, migraine, and, indeed, any other condition we might care to mention. On the other hand, it is quite another matter to assume that the co-existence of two conditions such as palpable evidence of alterations in the complicated nasal cavities and such symptoms as headache, neuralgia, various eye symptoms, means, therefore, that these nasal conditions are causing the symptoms. In the discussions over asthma there were those who assumed that this condition was always dependent on some alteration in the nose, the correction of which would cure the asthma. This was clearly but one way of stating that the relief from asthma was dependent on the skill displayed by the rhinologist in his efforts to discover and to eradicate the causative nasal conditions, many of which appear to most of us more imaginary than real. It is at once apparent that such an attitude places the treatment of asthma in a most unsatisfactory state since it must inevitably lead to a great deal of misdirected, uncalled-for surgery.

The conscientious rhinologist who has a proper feeling for the responsibility which should be his of guarding the field of medicine for which he is sponsor, cannot but deprecate a teaching which leaves open a portal for indiscriminate surgery. This responsibility falls the more heavily on the properly qualified rhinologist today because this special field has, in recent years, been flooded by men who, after a few weeks' or months' attendance at clinics in our cities, have taken to operating on the nose and throat without any of the more important and much more difficult training for making careful examinations and for determining proper indications for surgical interference. In recent years I am encountering an increasing number of patients who have been subjected to unnecessary operations, usually upon the ethmoid and sphenoid sinuses, where we find no reason for believing that pathology had existed in these regions, cases operated for the relief of such conditions as typical, hereditary migraine and eye or ear symptoms which it would seem no stretch of the imagination should rationally connect with nasal sinus trouble.

It is always important to remember that reasoning post hoc, propter hoc, is often very misleading.

*Presented before the Minnesota Academy of Ophthalmology and Oto-Laryngology, St. Paul, Jan. 17, 1924.

It is just this sort of reasoning that supports osteopathic or chiropractic treatments or the wearing of the hind foot of a rabbit for the cure of rheumatism. To illustrate—because a patient suffering from a mild attack of acute catarrhal otitis media complicated by a facial paralysis which latter disappears in due time after the mastoid has been exenterated is no proof that the operation on the mastoid had any beneficial effect, for, as we all know, such cases of facial paralysis usually clear up spontaneously quite promptly on the subsidence of the catarrhal otitis media.

Let us inquire into some of the questions which have more recently been raised in rhinology. One of the first of these is the so-called vacuum headaches attributed usually to the frontal sinuses. The frequent rather narrow passage from this sinus into the nose, particularly when the naso-frontal duct opens into the anterior end of the infundibulum, makes the closure of this passage from acute swelling of the mucous membrane a rather simple matter. It is assumed that the closure of this duct takes place and is followed by the creation of a vacuum in the sinus and the development of pain over the sinus from negative pressure without inflammation of the membranes lining the sinus and without the accumulation of fluid in the sinus. How such headaches are made persistent because of chronic hyperplastic changes in the underlying bone, particularly of the unciform, has all been elaborated with great detail. The occurrence of such vacuum headaches cannot be very common since I have never been satisfied that I have encountered a case where the hypothesis of a vacuum within the sinus offered the most plausible explanation. What shall we say regarding the occurrence of the type of headache attributed to vacuum formation with the so-called sensitive Ewing's point over the orbital wall and where the skiagraph demonstrates the complete absence of any frontal sinus?

There is another question which I have found difficult to answer if we accept the vacuum hypothesis for frontal headache, and that is the failure to find these headaches in cases of advanced hyperplastic ethmoiditis where the development of polypi completely closes the middle meatus. It would appear that in just these cases where we are most certain of the closure of the naso-frontal duct the vacuum headache must be the rule. As a matter of fact, I have never found headache of any type a frequent symptom of this condition. It is just cases such as these that question the validity of the whole theory of vacuum headaches. It would seem, moreover, from analogy with the obstruction to the Eustachian tube that the sinus would soon fill with a serous exudate, not a condition which in itself causes pain for it would not be a fluid under pressure, but a condition which would very quickly relieve any negative pressure. Sinus disease in which there is accumulation of inflammatory exudate within the sinus is a common symptom of sinuitis particularly when there exists an obstruction of the ostium. On the other hand, the occurrence of headaches independent of any palpable sinus disease is very much more common.

Some very interesting observations have been made on the relief of certain types of headaches by the application of cocaine or other analgesic agents, for example, to the region of the sphenopalatine ganglion. This phenomenon has been taken as an indication of the manner through which inflammatory disease of the nasal cavities produces involvement of the nerve trunk either by extension of toxins or of the inflammatory reaction through the bone or as the result of hyperplastic changes in the bone presumably setting up neuralgia through pressure on the nerve trunks. The fact that cocain passes through the nasal membranes and affects the nerve trunk or ganglion is no reason for attributing the same penetrating faculty to the products of inflammation. In support of this hypothesis for the local cause of these neuralgias, the great frequency with which the membranes of the nose become the seat of acute inflammatory attacks is pointed out. In addition, an effort has been made to associate certain chronic, rather impalpable alterations in the nasal mucous membranes with the etiology of these neuralgias on the theory that these alterations in the nasal mucosa indicate hyperplastic changes in the underlying bone which in turn effects an injury to the nerve trunk. All of this is very interesting and while it is perhaps not easy to disprove, we must all admit that it is even more difficult to prove.

I have recently been studying a case with the typical spheno-palatine neuralgia syndrome and where an application to the region of this ganglion just back of the posterior tip of the middle turbinated body is quickly followed by a cessation of the pain. The patient has perfectly normal nasal

passages so far as I have been able to discern, nor is she in any way particularly susceptible to attacks of acute rhinitis. Now are we to assume that there is some often quite impalpable local change going on in the nose in such cases that extends to and involves the nerves? Why is it not more probable that the cause of the spheno-palatine neuralgia is the same as the more usual cause of neuralgia occurring elsewhere, not a local cause at all, but some focus of infection, placed at a distance from the nerve affected? Such an explanation would seem more logical than to assume a purely local cause acting in a manner quite unique for these particular nerve trunks.

All of these conclusions have a bearing on the problem of handling such cases. If neuralgias of the spheno-palatine are the result of the same cause as a neuralgia elsewhere then our first aim should be to search out and eradicate any existing focus of infection and resort to the local treatment of the ganglion only when the neuralgia is not relieved by these other measures which aim to eradicate the original cause.

When we add to these pain syndromes from the nerves occupying the bony wall of the nose the question of the involvement of the optic nerve because of its proximity to sphenoid and posterior ethmoid cells, the rhinologist is indeed confronted by a serious and difficult problem. In the first place, the ophthalmologists assert that they are not capable of telling just what cases of nerve involvement are caused by extension of sinus disease. On the other hand, the competent rhinologist is rarely left in doubt about the diagnosis of a sinus disease where this is associated with a suppurative process. But we are confronted by the proposition that the sinus disease may be a hyperplastic non-secreting disease of which the skiagraph gives no clue and for which neither anterior nor posterior rhinoscopy can give us any tangible evidence, unless, perchance, we use a peculiarly brilliant light and even then the evidence is so impalpable that few will be able to recognize the trouble.

These propositions demand the most serious consideration from the rhinologist for if they are entirely correct then it certainly is incumbent on the rhinologist in most of such cases, at least of optic nerve involvement, to operate on the posterior ethmoid and the sphenoid. The operation must be undertaken not only in those cases where a suppuration in these cells can be diagnosed, but even when an absence of a suppurative process can be reasonably assured. This is, to say the least, not a very satisfactory position for rhinology. It is very much the same position in which rhinology found itself in the discussion over nasal reflexes in the eighties, for it opens the way, as did those discussions, to rather indiscriminate intranasal operations, with this exception, that the operations on the posterior ethmoid and sphenoid are much more formidable undertakings than the proposal for the relief of nasal reflex neuroses.

Let us inquire into some of the questions that arise in this problem of optic nerve involvement. In the first place, it is evident from the frequently demonstrated proximity of the optic nerve to the posterior ethmoid and sphenoid sinuses that inflammatory diseases of these cells might extend to injure the nerve. Do we not have an analogous situation when an involvement of the facial nerve develops in association with an otitis media? In the case of the facial nerve, it is apparently as often the extremely mild acute catarrhal process as it is the more severe suppurative disease that produces facial paralysis. Moreover, it is the acute processes that affect the nerve. Facial paralysis is never the result of the chronic, hyperplastic adhesive middle ear catarrhs nor is it often the result of chronic suppurative otitis media even where the hyperplastic changes in the mucosa and underlying bone are more conspicuous. Where it develops in chronic suppurative otitis media, it is usually the result of erosion of the canal walls by cholesteatoma or it is the result of sequestrum formation.

How these facts affect the problem of optic nerve involvement of nasal origin is quite obvious. Such involvement should be expected usually in connection with acute processes and not necessarily either the more severe processes. We should hardly expect nerve involvement from any chronic non-secreting hyperplastic process nor should we expect it to develop in the severe chronic suppurative process unless these conditions are associated with severe persisting headache indicating either retention or necrosis. The absence of nerve involvement in association with the chronic hyperplastic nasal process is quite in keeping with what we know of the bony changes in such processes. Beginning with the contribution of the English rhinologist, Woaks, attention has been directed to the bone changes in hyperplastic ethmoiditis. That such bone changes differ in any way from those that

result from periostitis in any other locality has not appeared. That these hyperplastic bone changes take place on the nasal aspect of bony walls cannot be taken as proof that similar changes occur on the opposite side of the bone, that is, on the orbital wall or the inner aspect of nerve channels. This is a problem still to be worked out.

When an optic neuritis occurs in connection with an acute sinuitis involving the sphenoid and possibly the posterior ethmoid is it desirable to subject all of these cases immediately to operative treatment? It has been urged in support of this procedure that some cases of optic neuritis proceed to recovery after the operation on the sinuses. But who can assure us that the improvement does not result in spite of the operation rather than because of this work? Do not many of these cases recover spontaneously on the subsidence of the acute sinuitis exactly as do most cases of facial paralysis that complicate the mild attacks of acute otitis media and that without any mastoid operation? Again, is not the recovery of the optic neuritis that occurs after an operation for draining the posterior ethmoid and sphenoid where we have clinical evidence of only a mild type of acute sinuitis quite analogous to the recovery of the facial paralysis in the mildest form of acute otitis media after there has been performed a quite unnecessary operation on the mastoid?

Gentlemen, I feel that all of these are pertinent questions which we should think over when we are confronted with a case of optic neuritis associated with an acute sinuitis.

In general, the logical conclusion would seem to be in acute cases of sphenoid sinuitis, the decision to operate should be made only after looking into the degree of nerve involvement on the one hand and the severity of the sinuitis on the other. Where either of these factors is severe, operate at once. When both are mild, do not be too hasty about operating since the probability is that there will be a spontaneous recovery in the course of a few weeks. When such recovery is not forthcoming, especially when there is a persistence of the sinuitis, the question of recommending the operation should be again seriously considered.

Now we come to one of the most serious of all the problems in connection with this question of optic neuritis and the occurrence of sinuitis. The ophthalmologist diagnoses a case of optic nerve involvement, the patient is referred to the rhinologist, who is unable to discover any palpable evidence of sinuitis or ethmoiditis. In spite of this negative finding, he is asked to operate on these sinuses because it may not be possible in all cases to positively exclude the occurrence of posterior ethmoid or sphenoid involvement. As proof that there existed an undiagnosed, undiagnosable spheno-ethmoid condition responsible for the eye trouble, it is pointed out that in some of these cases the optic nerve trouble subsides in time after the operation on the sinuses. But the matter is not so simple as all this, for in other cases in spite of the most radical operative measures, no improvement in the nerve takes place. Who shall say but that in those cases where improvement has followed an operation when no sinus disease could be discovered this improvement has not been in spite of rather than because of the operation.

There is another thought that arises in just such cases where the rhinologist finds no evidence of sinus disease. An acute involvement of the optic nerve even when this develops in the wake of an acute rhinitis, is no proof that such involvement occurs as the result of direct extension of inflammation or infection through the sinus walls. Only last week I had the opportunity of studying two cases, both occurring in young adult life, of acute neuritis of the eighth nerve. In one there was the history of a mild attack of coryza preceding the nerve involvement; in the other the only cause given by the patient was that she went out in the cold too soon after giving her head a shampoo. In both of these patients there was distinct evidence of focal infection both in the teeth and in the tonsils and the logical conclusion seemed to be that the latter was the important etiological factor in the development of the neuritis of the eighth nerve. The question naturally presents itself in acute optic neuritis when the etiological factor is obscure, even when the process is associated with an acute head cold but without palpable evidence of a sinuitis, may not focal infection be the important etiological factor? To my mind the two situations are quite analogous and it does not seem entirely logical to assume the existence of some impalpable, undiagnosable sinus condition as causing the optic neuritis when another explanation entirely different has to be assumed for the other. It has seemed to me that the importance of focal infection in the causation both of headaches, the sphenopalatine neuralgia, as well as of the involvement of the optic nerve

has been overlooked and that in the efforts to fix upon both of these conditions an etiology primarily rhinological, there has been a resort to hypotheses which are not only unproved, but are essentially unprovable, although one may not be able to disprove them.

These more recent problems that have come into the field of rhinology even though they may all eventually be put aside as having no great value have nevertheless already served a good purpose in stimulating a greater interest in rhinological work and have demonstrated in a most forceful manner the necessity of providing opportunity where men seeking preparation may be able to get something more than a smattering of operative technic while neglecting the more difficult and more important training in examination and diagnosis.

The conscientious rhinologist will guard with scrupulous jealousy the work in the field for which he is sponsor. He is not only anxious to avoid the risk of indiscriminate, unnecessary surgical work, but he is equally anxious to make sure that everything that is possible is being done for his patient. When such a serious condition exists as the loss of vision through an optic neuritis, the decision whether or not to operate should be reached only after consultation with the ophthalmologist, and, I might add, he should be ready to carry out any reasonable operative procedure even though he may be in doubt as regards the existence of a nasal cause.

NUGA-TONE

This is a nostrum sold on the mail order plan by a concern in Chicago called the "National Laboratory"—the latest name under which one Charles E. Cessna carries on mail order quackery. Nuga-Tone is described as the "Great Nerve and Blood Builder." It has been advertised in that class of weeklies sometimes described as the "cheap and nasty." The advertising does not tell just what is in Nuga-Tone. It does say that it is "rich" in iron and phosphorus, and also contains cascara and "nux." Further, one is told, "there are four other medicines" in Nuga-Tone. The death of a boy, three and a half years old, who had taken Nuga-Tone tablets in his parents' absence, is reported. When the death was brought to the attention of the National Laboratory, a formula was furnished which showed that each tablet contained 1/60 grain of mercuric chlorid, 1/60 grain of strychnin sulphate and 1/40 grain of arsenic trioxid. There is no law in this country which prohibits the indiscriminate sale of such dangerous nostrums. — *Jour. A. M. A., May 17, 1924, p. 1628.*

THE THIEF IN THE NIGHT—NON-INFLAMMATORY GLAUCOMA

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Stealthily creeping in, without outward visible sign or warning, to rob us of our most treasured sense, is the picture in his mind's eye, ever to keep before him, that every practitioner of medicine should have of simple, non-inflammatory glaucoma, for no more tragic affliction is the lot of man. Generally, one might say most frequently, unrecognized until well advanced, the most precious time for its combatment is only too often used up by the optometrist, and others untrained in ocular pathology and clinical ophthalmology, in repeated change of glasses—given with the opinion that the patient "is becoming far-sighted" or that "it may be an oncoming cataract," et cetera. And finally, only too late for help and after optic nerve atrophy is well on its way, the patient consults the oculist, whose sad and painful duty it becomes to tell that nothing can be done; and the unfortunate person must drift into the long night of blindness.

"Early recognition means everything. Late recognition or wrong diagnosis means loss of everything" (Fuchs). Although primary glaucoma, according to Fuchs, constitutes 1 per cent of all eye diseases, how extremely infrequently the general practitioner recognizes it or even suspects it only the well-occupied oculist is conscious of, and this, too, in cases of the inflammatory form. Iritis and iridocyclitis, in the presence of an inflamed eye, seem almost always thought of, and that diagnosis is generally made in most cases even if the patient is fortunate enough to be spared the catastrophe of having atropin put in his eye. How much more rarely, then, the non-inflammatory form, without a single outward objective sign, is even suspected, can well be imagined. Just such cases are the ones which, during the very time that something could be done for them to halt the dreadful malady, are glassed and reglassed by the optometrist and others without special training, only to lose steadily the remaining vision they possess. These cases, too, as we all know and are sad in the realization, go undiagnosed through the hands of careless and superficially working oculists,—the kind of refractionists who are satisfied with less

than normal vision in their patient without finding the reason why; who pass over accommodation inability in their patient without the compatibility of age or hyperopia; who are in too much of a hurry to take the field of vision when it is indicated; and whose dark-room observation is less than a careful, thorough, and searching examination of the anterior segment of the eye by oblique illumination and loupe, and the posterior segment of the eye (including lens, media, peripheral retina, macula and nerve-head, its color, size, margins, elevation, cupping and vessels and their behavior) with ophthalmoscope. Just such work has belittled scientific refraction, than which no branch of ophthalmology is capable of requiring more high training, good balance and sound judgment; this type of man's refraction takes no longer to do than the optometrist's, and in reality is not worth any more, and is responsible now for the growth and prominence of the optometrist and his kind in our midst.

A fact all oculists must ever impress upon themselves is that glaucomatous cupping with atrophy is too late for help. Cupping prior to atrophy is what we strive to find and it is our business to recognize it.

There are three types of cupping of the nerve-head: (1) physiologic; (2) atrophic (ordinary optic nerve atrophy); (3) glaucomatous.

Physiologic cupping is partial cupping, never total cupping of the entire breadth of nerve-head, nor deeper than the lamina cribrosa in its normal location. Any depth of the physiologic cup is due to its depth in the plump, heaped-up nerve-fibers, and the vessels do not emerge at the margin of the disc. The lamina cribrosa may be seen at the apex of the funnel but not across the whole width of disc at the floor of the cupping. On the other hand a total cupping of the entire breadth of nerve-head from margin to margin is always pathologic and must be either atrophic cupping or glaucomatous cupping.

The cupping of atrophy of the optic nerve, or so-called atrophy cupping, is a shallow, flat, white-bleached cupping never deeper than the lamina cribrosa in its normal location, and the atrophy is present before the cupping takes place; in fact, the shrinking or atrophy of the nerve-fibers makes all the cupping there is, with fenestrae of the lamina cribrosa exposed as a result of shrinkage.

The cupping of glaucoma, rather shallow at first, is deeper than the normal location of the lamina cribrosa; is caused by its recession under intra-ocular pressure; and is there before atrophy of the nerve-fibers takes place; in fact, the after-coming shrinkage of nerve-fibers in the already existing cupping adds considerably to its depth. It is just at this stage where we as oculists should be on the job; that is, recognize the cupping (recession of the lamina cribrosa) before atrophy takes place and while the nerve-head is yet a healthy pink and the fibers plump; before the blue-green color appears and before the shrinkage adds to the depth of the already existing excavation and the exposure of fenestrae of the lamina cribrosa. After these are present it is too late for help. At this stage, before the healthy pink color fades and before the lamina cribrosa fenestrae are exposed by shrinkage, the proximity of the vessels to the very edge of the disc should immediately put us on our guard. Later the characteristic bending over and disappearance of vessels at the edge takes place, but this is generally found after the added depth of the excavation results from shrinkage.

Even in the presence of a well-defined glaucomatous change in the nerve-head in a hurried, superficial inspection, especially by indirect ophthalmoscopy, it is very easy to miss the telltale behavior of the vessels, which, reappearing at the bottom of the excavation, upon close examination, can be seen to have a different hue, and by paralactic displacement can be seen to be at a marked difference in depth. This, I repeat, can be easily missed except in detailed inspection by direct ophthalmoscopy, especially while the nerve-head is still pink in health and before atrophy has arrived.

Contracted arteries and slightly distended tortuous veins should immediately arrest the attention of the observer, for at this stage the recession of the lamina, prior to atrophy, does not include as yet a decrease in vision, and the time is even here at hand, by proper medical and surgical relief, to arrest and avert the atrophy which does mean loss of vision. Even in the presence of large, unmistakable pathologic cupping good vision and uncontracted fields may be found.

Of course the exception to this can be found in acute fulminating inflammatory glaucoma, where the pressure comes on so suddenly and acutely that destruction of life in the nerve-fibers takes place even before the lamina has had time to yield and recede. In this form of disease very often, if not

too prolonged, considerable recovery of vision may be found, but that it has suffered can be demonstrated in careful field-taking, by the presence of sectors or gaps reaching even to the macula or normal blind spot.

Ordinarily, then, estimation of glaucomatous damage can best be made not by the excavation but by the color of disc (pink color of health or gray-white of atrophy) and by the size of the arteries. The classic halo around the excavation is due to sclerosis and pressure-atrophy of the choroid; but this, of course, is a late, too late, finding.

Glaucoma—that is, inflammatory glaucoma—has been known from antiquity, and all the old writers have associated it or thought of it in connection with gout and arteriosclerosis. Simple, or non-inflammatory glaucoma, was never known or thought of in connection with the known (inflammatory) glaucoma until the advent of and since the progress of our knowledge from the use of the ophthalmoscope. Mueller first demonstrated glaucoma cupping anatomically in 1856.

Intra-ocular pressure and tension were first associated with glaucoma by Mackenzie and by von Graefe. Mackenzie relieved it by repeated paracentesis, but without permanent result. Von Graefe discovered, merely by coincidence, that iridectomy relieved it and in most cases with permanently good result, and this discovery proved to be one of the greatest advents in the history of ophthalmology. Von Graefe's declaration that the "essence of glaucoma lies in the increase of intra-ocular tension, from which all symptoms can be deduced," really stands unchallenged today and is the universally accepted idea of glaucoma.

Two chief divisions of glaucoma are: (1) primary and (2) secondary. Primary glaucoma comes on without any known antecedent disease. In this group are the inflammatory and the non-inflammatory affecting both eyes, generally one after the other in its start, but ultimately both. This class is the real glaucoma or the pure glaucoma. Secondary glaucoma is really merely a complication of tension in an already diseased or injured eye, resulting from the interference of exit of the intra-ocular fluids from the eye, and affecting only the eye in which the pre-existing disease or injury is present. It is merely an accessory and would not have come on except as a result of such disease or injury. Secondary glaucoma is, of necessity, mostly an inflammatory glaucoma.

The common conception of the entrance of the optic nerve as through a round hole or opening in the sclera is entirely incompatible with a correct understanding of the pathology of glaucoma. There is not a gross gap or gross opening in the sclera at the point of entrance of the optic nerve. The scleral fibers are merely pushed aside like the slats of lattice work, offering a multiplicity of minute fenestrae through which the fibers in small groups pass into the eyeball, the so-called lamina cribrosa. Nevertheless, though the sclera is not grossly perforated, this spot is the one vulnerable spot in the event of intra-ocular tension, and this lattice work is the first to recede under pressure. The neighboring resistant sclera surrounding this lattice-work area affords a non-yielding ring over which the fibers, in their spray-like arrangement, pass in their distribution to form the retina. These fibers are broken and pinched by pressure until atrophy supervenes, which can be likened to a hernia which is strangulated in its unyielding hernial ring. Over these precipitous edges or eaves the vessels from and to the nerve-trunk are forced to take their course, disappearing over the eaves like drain pipes, and reappearing at the bottom of the cup or excavation. Late in the atrophy, through shrinkage of nerve-fibers, these small fenestrae of the lattice work can be seen, too, at the bottom of excavation. With the atrophy of the optic-nerve fibers which go to make the retina, of course loss of vision comes on; central as well as peripheral vision. The nasal field is first to suffer by reason of the temporal side of the retina being made up of the longest fibers from the nerve, and farthest from their center of nutrition, and consequently the most vulnerable in degeneration.

In order to present a comprehensive picture of glaucoma, it will be best to describe the acute inflammatory type, although not the type indicated by the subject of this paper, nor the type intended to be brought out in this discussion. Yet, as a starting point, all forms can be best visualized by shading up and down; up to the so-called malignant type which can strike suddenly and, unrelieved, can go on to complete and permanent blindness through a short and stormy course; and, shading downward, to the type which is the subject of

this paper—viz., the treacherous, unsuspected, non-inflammatory type—without outward sign to warn the patient or to indicate to friends, associates or even the medical man, the insidious, slow but sure oncoming blindness. For this reason, to repeat, it is seldom recognized until the fading vision from atrophy is upon us and beyond any medical or surgical help.

Promodes: A complaint of smoky vision and dull frontal headaches lasting a few hours, which may recur at intervals of a few days or even weeks or months, and which may be mistaken for presbyopic symptoms if the patient is in the forties, may be accepted as due to strain and symptoms of presbyopia alone. Recession of the accommodation near-point might not even put the oculist on his guard with these patients. An added complaint of halo or rainbows around lights with, ordinarily, a frequent necessity for increasing strength in reading glasses, jolts the eye-man into "attention" but means little or nothing to the optometrist or others untrained in the specialty. Generally these symptoms disappear during sleep and rest, and in the very mild inflammatory type these periodic symptoms may not increase much in severity for years, but all this time the optic nerve-head is leading up to atrophy. As these periodic spells recur, vision slowly recedes, accommodation lags and the victim is given stronger glasses. Usually, however, sooner or later an acute flare-up supervenes, after some gastro-intestinal disturbance, mental emotion, obstinate constipation, et cetera, and is ushered in by violent pain in the ears, the teeth, the head, and the region of the eye. Sleeplessness, anorexia, and maybe nausea and vomiting, are only too often accepted as acute grippe with neuralgia in the head. Then vision in the affected eye begins to fail; mere objects are only discernible; and at last the eye is recognized as the seat of trouble. Edema of the lids, chemotic conjunctiva, steamy cornea insensitive to the touch, with shallow anterior chamber and muddy, green iris, dilated, immobile pupil of peculiar green-gray color, oval or eccentric, and an obscured fundus and stony hard eye, complete the picture to the eye-man.

After a day or two or three, more or less, with treatment and sometimes without, the symptoms abate, tension recedes, the eye returns grossly to its usual appearance, vision seems to be restored, and patient goes on relieved for a time, sometimes for a long interval, sometimes for a short interval, and the attack is repeated. After repeated attacks, however, return of tension to normal is impossible as a result of pathologic changes, and the eye goes into a chronic hard, painful and blind eye. Even though recovery seems, grossly at least, complete between attacks, close examinations show vision to have suffered, and it declines steadily, and the gray-slate-color-change in iris with exposed pigmentary layer at pupil edge, dilated anterior ciliary veins and porcelain-white sclera with sluggish pupil and optic-nerve cupping, make the classic picture. The degenerative corneal processes, scleral ectasia, calcareous lens and atrophy and shrinkage of the globe, form the picture of terminal, chronic, absolute glaucoma.

The above description is that of the entire course of the average primary inflammatory glaucoma, and though it starts in one eye and may long antedate its advent in the second eye, only too often the same process is repeated in the other, for it is regarded as a bilateral disease of advanced life in people of fifty to seventy years of age, most commonly of all among people of the Jewish race.

Shading down from the above picture, types of all grades are found, where symptoms are regarded as "billiousness," "neuralgia," "rheumatic headaches," et cetera; and we gradually come to the least manifest, but at the same time the most fatal form, the simple non-inflammatory form, "the thief in the night," the type that I heard one of the greatest of teachers of ophthalmology say would be about the only thing that, afflicting him, would make him a suicide.

The chief condition calling for differential diagnosis in the inflammatory type is, of course, iridocyclitis. Here we have generally a contracted pupil, but just there is the necessity for caution because glaucoma frequently complicates a former iridocyclitis with iris contracted down and held by adhesions, or even complicates an existing iridocyclitis; and the uncertainty in which we find ourselves as to the proper medication, atropin or eserin, puts us in the position of a person with a torch in one hand a fire extinguisher in the other, undecided which to use, generally concluding in the better judgment to use neither. In fact, the eye of every patient in middle life, or beyond, must be always approached with care and caution in the use of cycloplegics or even mydriatics. The instillation of even such a short-acting mydriatic as cocain has many times precipitated an attack of tension in eyes

that have previously never been known to have any disposition toward glaucoma. A careful eye-man will constantly guard against such ambush by using pilocarpin after the use of cocain, even for the removal of foreign bodies, in patients of such an age. An iridectomy in one eye has been frequently known to precipitate an attack of glaucoma in the opposite eye, adding to the distress of patient and attending oculist.

In general it should always be borne in mind that hyperopes with small, short eyes are chiefly the subjects of such tendency, while myopes with long eyes are rarely so disposed. In the use of all cycloplegics, patients of all ages should be close enough at hand for observation. Once in the experience of the writer acute inflammatory glaucoma was precipitated in both eyes of a young girl of twenty-two years of age by the use of homatropin. Fortunately she was under control, and within a few hours following the onset, vigorous treatment to combat the condition was instituted and proved effective; but such an experience is enough to last a lifetime. In my experience tension, too, has several times developed in cases of iridocyclitis after the use of atropin, where paracentesis and even iridectomy had to be resorted to to meet the emergency of sudden high tension.

It is an unwise and altogether unsafe practice to have patients living out of town continue to use atropin when they are out of reach and observation, and I know of patients who have suffered total and absolute blindness during the interval betwen trips to the oculist in the city.

In marked contrast to the inflammatory glaucoma described above, with abundant objective as well as subjective symptoms, is the simple non-inflammatory type, where the subjective symptoms are so slow and insidious as not to arouse even the patient until the effect of atrophy comes on, and where scarcely a single outward objective sign is manifested even to the alert and observing oculist. His contracting field is often not discovered by the patient because his central vision usually remains very good. The distended anterior ciliary veins around the cornea may be enough to arrest his attention, but even this is not prominently present until the condition is of rather long duration. Prior to the invention of the tonometer by Schoitz, of Christiania, glaucoma simplex was not known to be the result of raised intra-ocular tension, for the finger test by palpation could not determine it. In these cases only repeated tonometer readings, sometimes repeated two or three times a day, are enough to establish the fact.

Just as slight continuous pressure in the dilatation of a uterine cervix by the water-filled placental membranes is more effective for complete dilatation in that structure than forceful instrumental dilatation of short duration, likewise the continuous, constant low-pressure of the slightly raised intra-ocular tension of simple non-inflammatory glaucoma is more fatal and sure of excavation and atrophy in the optic nerve-head than the high-pressure of a violent inflammatory glaucoma attack of short duration. Moreover, an acute attack is so adequately relieved by a properly performed iridectomy that since von Grafe's institution of that procedure the inflammatory type has largely lost its terrors to the ophthalmological world. On the other hand recognition of the simple non-inflammatory type is almost always so long delayed that almost any and all procedures, surgical and medical, are dismally ineffective and of no avail. As a consequence, announcement of the presence of the simple non-inflammatory type in a given case has the effect of a funeral pall upon us all, and utter hopelessness and despair engulf even the most optimistic youngsters as the result of their reading and teaching, and surely stirs no hope in older men of years and experience. Nevertheless I believe, and I am sure that all of us believe, that the pathology of all forms and types of glaucoma is fundamentally the same, and that if the simple non-inflammatory type could only be recognized as early, antedating atrophy, as the inflammatory type, its relief could and would be just as sure and effective as in the latter.

Let us all, then, be alert in the recognition of the pre-atrophy stage, the stage where the recession of the lamina cribrosa is to be made out on careful inspection; where the cupping can be seen to reach from margin to margin, but before the healthy pink color fades and before the atrophy coming on additionally deepens the excavation and exposes the fenestrae in the lamina as a result of the shrinkage of the fiber bundles. If we are alert at this point and proper medical and surgical relief is instituted, there is no doubt that the malady can be arrested as certainly as in the inflammatory form.

Sometimes the simple form flares up into the inflammatory. It would be fortunate if it always did, for then it would be recognized. For, while the

inflammatory form of primary glaucoma is almost always a disease of middle or advanced age, the simple non-inflammatory type only too often claims its victims from among the comparatively young adults, adding to the tragedy.

The inflammatory reactions of iridocyclitis and glaucoma are different in that the former, when severe, is attended with plastic exudate, while that of glaucoma is distinctly the serous or water-logging process of strangulation. In the purely glaucomatous inflammation of the eye hypopyon or pupillary exudate is not found, a very important point of differentiation between the two diseases. The clouding of the cornea, too, is caused by this same water-logging condition and quickly disappears upon relief of the strangulation, which is not the case in corneal inflammation or keratitis.

The essential condition in all types of glaucoma to be borne in mind is the interference or blocking of the exit of intra-ocular fluid from the eye at the angle of the anterior chamber, be it as a result of a swollen or thickened lens, a congested and enlarged ciliary body that presses the root of the iris forward to the cornea, or to a general hyperemia and edema of the whole intra-ocular tissue, or to the presence of exudate or disintegrated blood elements smearing over and clogging the filtering structure of the ligamentum pectinatum and Schlemm's canal. The effect is the same, viz., a disturbance of balance between the incoming and the outgoing fluids of the eye resulting in pressure and strangulation of retineal structures which eventually atrophy and perish.

The only communication from the posterior chamber to the anterior chamber of the eye is through the circumlental space, and in case this space is impinged upon by a disproportionately sized lens as a result of swelling, the lens acts as a valve seated against the ciliary processes and iris base. The increasing pressure thus pushes these structures forward, jamming the angle of the chamber and shutting off the exit of intra-ocular fluid from the eye.

The most favorable factors for this condition are a small eye, shallowness of anterior chamber, a large ciliary body and a disproportionately sized lens. Towards all of these we have a tendency as age advances in the originally hyperopic eye, and in many instances only the added peripheral thickening of the iris when dilated is necessary to precipitate the blocking of exit for the fluids, and the strangulation sets in.

Iridectomy for relief of glaucoma should always be done as early as possible by making section well into the sclera, rather than in the cornea, and should be as broad as possible and to the very root of the iris. Where regional atrophy exists in the iris, the iridectomy should be done in the least atrophic part, for in this region it will be more effective in opening the angle of the chamber. The various forms of sclerotomy, including trephining, have not proved as dependable as the broad iridectomy, and even in the simple non-inflammatory form the latter would, no doubt, be the most dependable if it could be done in early recognized cases. The action and benefits of myotics in the medical treatment, of course, are to be seen in the drawing away of the peripheral folds of the iris at the angle of the chamber such as takes place in active contraction of the iris. Eserin and pilocarpin can be efficacious only where the iris muscles are capable of contracting, surely not where atrophy of that structure makes such contraction impossible. The action of myotics should be welcomed only as a tide-over and not as a substitute for proper surgical relief, for when their effect wears off the tension is likely to return. The liberal use of myotics before operation is a great help. Tension may be partially relieved, anesthesia may be more effective, and the operation is rendered far easier in performance.

Infantile or juvenile glaucoma, hydrophthalmus, buphthalmus or ox eye, is distinctly a disease of childhood, and like glaucoma in the adult is also caused by intra-ocular pressure, due to an obstruction of the avenue of exit of the intra-ocular fluids; but the pathology primarily is, in all probability, a congenital absence or relative absence of the normal drainage passages known as the canal of Schlemm, or an impervious ligamentum pectinatum, and is not due to obliteration of the angle of the anterior chamber from encroachment of the adjacent iris, as in the ordinary adult type of glaucoma. During the years of early childhood the sclera and cornea are relatively soft and yielding, and as a result give way before the increased intra-ocular tension. The eyeball enlarges to tremendous size; hence the popular name, ox eye. The outer supporting coats, almost alone, take part in the enlargement, and as a result the intra-ocular structures, especially the lens, are proportionately small. The

sclera of characteristic bluish-white, the strikingly large cornea, deep anterior chamber, the shreddy, tremulous iris and the prominence of the globe, make diagnosis easy. Examination of the nerve-head will disclose the glaucomatous cupping. The process can go on to enormous size with ultimate total blindness, or, as is frequently seen, it stops spontaneously with some remaining vision, but the eye remains disproportionately large and disfiguring. It may be unilateral, but usually is bilateral. The lens is often loosely held by its ligament, which is either partially or entirely ruptured, and becomes cataractous, and drops to the bottom of the vitreous chamber.

SAN-I-SAL

Newspaper advertisements have appeared during the past month or two advising the obese public of "A Guaranteed Way to Take Off Two to Five Pounds in One Bath." The product advertised is "San-I-Sal, the Pine Hot Springs Bath." It is sold by the San-I-Sal Laboratories, Washington, D. C. The advertising claims are typical of "patent medicine" obesity cures. According to the specifications of a patent granted by the U. S. Patent Office, the "invention" is said to relate to "a composition of matter for producing a medicated bath of particular value in the treatment of obesity." The "composition" is stated to be: epsom salt 90 per cent, baking soda 2 per cent, table salt 5 per cent, Canada balsam 2 per cent, oil of pine needles 1 per cent. The specifications also declare that the substance used in the bathing water has been found to afford "great relief in cases of inflammatory rheumatism and ailments of a similar character." That a mixture of epsom salt, baking soda and table salt with a small quantity of Canada balsam and oil of pine needles should be granted a patent by the U. S. Patent Office as a new and useful invention, makes one wonder just how much intelligence on medical subjects there is displayed by the Patent Office. —*Jour. A. M. A., May 31, 1924, p. 1800.*

LIPOSAN

According to the advertising, Liposan is "a Vegetable Lipoidal Solution" which has chaulmoogra oil as its "medicinal element." Liposan is claimed to be "indicated" in "Abscesses," "Acne," "Anemia," "Arthritis," "Agitans," "Boils," "Bubo," "Cancer," "Chorea," "Cystitis," "Eczema," "Erysipelas," "Furunculosis," "Herpes," "Hemiplegia," "Ivy Poisoning," "Myalgia," "Neuralgia," "Neuritis," "Peritonitis," "Pyorrhea," "Infantile Paralysis," "Pneumonia," "Rheumatism," "Synovitis," "Syphilis," "Salt Rheum," "Tubercule," "Tonsilitis," "Ulcerations (Indolent)," "Varicositis." The proprietors, Hoffman and Hicks, have not requested an examination of Liposan by the Council on Pharmacy and Chemistry, and so far the A. M. A. Chemical Laboratory has not examined the product. A physician will be justified in his refusal to accept the unproved claims of the manufacturer. He might be justly criticized were he to administer intravenously a product which, so far as he knows, is unstandardized and of wholly questionable value.—*Jour. A. M. A., May 3, 1924, p. 1462.*

MENTAL RE-EDUCATION IN THE SERVICE OF MEDICINE

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Psychotherapy has always been intrinsic to the art of medicine. In the distant past, healers had their charms and amulets. The medicine man of our own American Indians, as of many other primitive peoples, had a psychotherapy, which was one of fright and fury. The fine type of general practitioner we have heard idealized through the past two centuries was the embodiment of a beneficial mental influence, which comes legitimately to the aid of scientific medicine in its war on both organic and functional disease.

With the different stages of medical progress there has been a corresponding amount of insight into the mental factors of sickness, and varying therapeutic methods for dealing with such manifestations have accompanied medical development. During the last twenty years the knowledge of the nature and causes of "nervous" disturbances has been revolutionized. The war neuroses have recently contributed to a general interest in such conditions. From this, and from many other sources, psychotherapy has gained a more intelligent base of operations. At present, among the most effective measures it employs is that of re-education; this signifying the giving to the patient knowledge regarding his own make-up, his moods and his motives, and likewise training him, as an individual, up to his maximum of what Dr. Adolf Meyer has aptly expressed "constructive composure."

The older order of treatment of the minor functional troubles presented a great range of variation. Sometimes an exhortation, or clapping of the unfortunate "neuropath" on the back, was followed with a tonic or with the assurance that nothing was wrong. On the other hand, there was considerable urging the patient to get well solely by dint of will and conscience, and without any knowledge of himself. Or frequently the physician, in paying careful attention to a rather unimportant physical abnormality, fixed a neurosis on an organ that had hitherto largely escaped the patient's notice. The doctor thus became, professionally, a factor in the production of a more chronic disability. It often happened that the physician himself summarily condemned the patient, ignoring those phases of

the situation which might be subject to improvement. A spirit of denunciation made the patient on the defensive and apologetic from the start. Sometimes this assumed the form of an exaggerated hypochondriasis, or self-depreciation, with real bewilderment. On the other hand, many times it bred an over-compensatory attitude of belligerency and dissatisfaction with the physician, to the detriment of the profession as a whole. It also provided the basis for the development of various present-day cults. These cults flourish on the emotional ills of the people, aided and abetted by the failure of the medical profession to meet psychic as well as physical needs of patients. Fortunately, better methods are gradually superseding these unsatisfactory forms of therapy, which were born largely of ignorance. No adequate teaching of medical psychology had been given in the medical schools. The physician's ignorance of psycho-dynamics, plus the pressure of his work, could not help but breed a most discouraged state of mind medically. Even now, in the gradual development of medicine, one too often finds the physician giving more time to a trivial physical disability than to a complex personality disorder. The patient himself is making strenuous, if unsuccessful, efforts to understand himself and needs the help of one who might be called an intelligent personality interpreter, who can make the patient himself understand the balancing, as well as the conflicting mental factors present in his make-up.

A physician who honestly devotes effort to analysis of personality is able to offer definite practical help to harmonious readjustment for living. With the sincere effort of the physician to face the patient's life situation, a corresponding ability comes to the patient, who previously had feared to meet reality. In fact, the overdeveloped measures of defense, such as hypochondriacal complaining, defiance, and doubt, are born of fear, and when courageously challenged are susceptible of control.

At the basis of any sound psychotherapy there must be specific insight into the condition. The well-known English neurologist, Buzzard, says, regarding this: "Born of the needs of the consultation room, within the past twenty years the science of history-taking for physical and later for mental disorders has been assuming its appropriate importance. On the physical side this has been accepted without demur, but on the mental side it has been met with a volume of resistance which only time and education are slowly breaking down." As one of our own leaders, Dr. White, has expressed it, "The method of estimating the patient's condition by what he is pleased to volunteer regarding it, has been found increasingly less satisfactory. Conscientious physicians are no longer content to take as final the patient's account of his discontent, his nervousness, sleeplessness, or general indifference, any more than they are willing to let a cough go uninvestigated."

With a proper inquiry into the causes underlying a "nervous" upset, there follows, in the logical treatment of the condition, a systematic enlightening of the patient with regard to his own make-up, and training to efficiency.

The process which marks a therapeutic change is a very dynamic one and alive with the keenest of life's intensities. The term "transference" is serving a useful purpose at the present time in describing that part of the process which deals with the personal relationship of patient and doctor. The physician forms a buffer between the patient and the world, and is the first recipient for new formed attitudes of mind. There are infantile habits to be changed, and poorly conditioned reflexes to be replaced by new ones. The method of attack has to be definite; no generalities or platitudes will be of use. While the physician is oftentimes in a passive rôle, yet ultimately his rôle must be an inquisitorial one. He has to be quietly on his guard, ready to enforce his points at the moment required, remembering, however, that much brilliant conversation is useless with hysterical or neuropathic patients; they are interested in themselves, not in another person. Through "therapeutic tact, rapport, experience," or what you will, the physician is able to give the patient a new moral balance, a broadened sense of obligation, or, in other words, a practical philosophy of life, suited to his abilities.

In this treatment of the individual, rather than the apparent disease, and in this temporary domination of personality over personality, oftentimes really remarkable reorganizations of lives can be effected. Unhappy, maladjusted people learn to drop their frantic, panicky, misdirected struggles, through which they are fleeing from their weaknesses and handicaps. With increasing success they become able to control their emotions and activities, and enter into normal social relations again, gaining the capacity to maintain a healthy, active attitude in the face of difficulties.

From time to time, physicians specializing in this type of work have put forth some constructive idea that they have elaborated in the process of re-education of patients. Recently two physicians, at different ends of the country, whose time has been devoted chiefly to psychotherapy, have each published a book containing suggestions of value regarding the process of personality reorganization. The reference is to Dr. Jackson of Pasadena, California, and Dr. Gehring of Bethel, Maine. The titles of their books are, respectively, "Outwitting Our Nerves" and "The Hope of the Variant." These books have in common the idea that intelligent self-knowledge given to patients is a powerful help in promoting their reinstatement to social efficiency, and that people can, in a large measure, choose the moods, emotions and activities that go for healthy and happy living. Dr. Gehring's work was preluded several years ago by Herrick's "The Master of the Inn," a book written from a patient's experience and point of view.

In Dr. Gehring's present study, the personality of the variant, or potential neuropath, is said to be within the wide range of nature's normal zone. The author shows that the variant can obtain a calm state of mind and cultivate a sturdy, optimistic attitude toward obstacles. Such a one must learn that many of his worries and chronic fears are born of a faulty habit of comparing himself, detrimentally, with others, which can be replaced by the quieting feeling of security that he only has to know and be loyal to the best in himself. One particularly suggestive point Dr. Gehring has studied out is that obsessive concepts come through the channels of feeling, and that they can be modified and often made to disappear by the process of getting them formulated or crystallized into words.

Dr. Jackson's book deals more explicitly with instincts and emotions, which she explains are the well-springs of behavior. These lie deep in each individual's personality and are bound up with much custom, tradition, and a variety of thought processes called rationalizations; the purpose and result of these latter being to justify, on supposedly intelligent grounds, the individual's instinctive course of action. One secret of self-control is in adjusting the receptive portions of the instinct mechanism, so that the threshold is less egotistically sensitive than is that of a neurotic. This process can be made possible through re-educational effort. She speaks of what "vital importance it is to society that its citizens should be taught to solve their inner conflicts and keep well; at the top notch of vitality with well digested food, well slept sleep, well forgotten fatigue, and well used reserve energy." She speaks reassuringly of the day of the long term sentence to nervousness being past. The book has done, and is still doing, good work in the hands of large numbers of patients.

Throughout present day medical literature there are innumerable short articles on the same general subject. The appended bibliography, used in the preparation of this note, gives an indication of how widespread are the tributaries of this current medical development.

In concluding, the broader implications of the subject may be mentioned. Re-education is, in reality, but one aspect of that larger problem of education which faces each one of us. Progress is accomplished through specialized effort becoming more readily available in the service of different fundamental obligations confronting each member of the "responsible generation." One does not need to find himself a professor of medicine, or such an exalted being as a member of a committee on medical education, or even a representative of a special branch of medicine, to take note of such considerations; for the challenge comes to each physician, parent, teacher and friend, to do what he can to further honest and ungarnished self-knowledge and purposeful social accomplishment.

BIBLIOGRAPHY

1. Ball, C. R.: Psychotherapy. New York Med. Jour., 1921, cxiv, 580-583.
2. Bramwell, R.: Psychotherapy in general practice. Edinburgh Med. Jour., 1923, xxx, 37-59.
3. Buzzard, E. F.: Psychotherapeutics. Lancet, 1923, cciv, 330-332.
4. Buzzard, E. F.: Some aspects of mental hygiene. Ment. Hyg., 192, vi, 449-462.
5. Gehring, J. G.: The hope of the variant. Chas. Scribner's Sons, 1923.
6. Hamilton, A. S.: Need of psychopathic hospital at University of Minnesota, Minn. Med., 1922, v, 683-686.
7. Harnsberger, S.: The neglected latent faculty. Virginia Med. Month., 1922, xlix, 397-398.
8. Head, H.: An address on the diagnosis of hysteria. Brit. Med. Jour., 1922, i, 827-829.
9. Jackson, Salisbury: Outwitting our nerves. Century Company, 1921.
10. Jelliffee, S. E.: Some notes on "transference." Jour. Abnormal Psychol., 1914, December-January.
11. Johnson, Alice E.: What is a "nervous breakdown"? Ment. Hyg., 1921, v, 784-790.

12. Maeder, A.: Sur la psychotherapie. Arch. f. Neurol. u. Psychiat., 1922, x, 100-112.
13. Martin, C. F.: Psychiatry and internal medicine. Canad. Med. Assn. Jour., 1920, x, 827-834.
14. McPhedran, A.: Importance of the emotional or physical nature of people in general in practice of medicine in its widest sense. Canad. Med. Jour., 1923, xiii, 82-84.
15. Solomon, M. L.: Make-up of neurotic. Illinois Med. Jour., 1922, xlii, 473-475.
16. Putnam, J. J.: Plea for study of philosophic methods in preparation for psychoanalytical work. Jour. Abnormal Psychol., 1911, vi, 249-264.
17. White, W. A.: Extending the field of conscious control. Psychoanalyt. Rev., 1920, vii, 148-158, or Mental Hygiene, 1920, iv, 857-866.
18. Williams, T. A.: Hyperemotivity: Its causes: Their permanent removal. Boston Med. and Surg. Jour., 1921, clxxxv, 477-478.

ZINC-BOROCYL NOT ACCEPTED FOR N. N. R.

The Council on Pharmacy and Chemistry reports that, according to the Al-Sano Chemical Products Co., Zinc-Borocyl is a definite chemical compound. The label of the trade package gives "boridiorthotic oxybenzoic acid zinc" as a synonym for Zinc-Borocyl, but it also declares that the product which is marketed is not the substance to which the name "Boridiorthotic oxybenzoic acid zinc" is applied, but that "the contents of this bottle represent a concentrated solution" of this product. From the information furnished the Council and the statements on the label and in the advertising, it may be concluded that "Zinc-Borocyl" is a solution containing the so-called zinc borosalicylate in undeclared amounts. Its exploitation is essentially an attempt to introduce an old German nostrum—Mucosan—to American physicians under a new name. Zinc-Borocyl is offered as a germicide, antiseptic and astringent, and is recommended for a host of conditions. The Council declared Zinc-Borocyl inadmissible to New and Non-official Remedies because (1) its composition is not correctly declared; (2) it is an unoriginal preparation marketed under a proprietary noninforming name; (3) the recommendations for its use are not upheld by acceptable evidence, and (4) the available information fails to show that the product claimed to be zinc borosalicylate has any advantage over established zinc salts.—*Jour A. M. A., May 24, 1924, p. 1712.*

FERRASSIN NOT ACCEPTED FOR N. N. R.

The Council on Pharmacy and Chemistry reports that Ferrassin is marketed by Robert Wollheim in the form of tablets and capsules. They are said to be composed of "Vegetable Iron," "Peptonized Iron and Manganese," "Plant Albumin," and Milk Sugar. No information is furnished in regard to the composition of "Vegetable Iron" or of "Peptonized Iron and Manganese." Ferrassin was declared inadmissible to New and Non-official Remedies because its composition is secret and it is marketed with unestablished and unwarranted claims.—*Jour. A. M. A., May 24, 1924, p. 1712.*

THE USE OF CONCENTRATED FOODS IN THE FEEDING OF INFANTS AND CHILDREN *

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In the past few years far-reaching investigation has been carried on in the feeding of infants and young children with concentrated food. Such feeding, however, does not constitute a new procedure; for foods of high caloric value which could be given in relatively small amounts have been used for years.

However, it is not until a comparatively recent date that any food other than the most dilute mixtures of milk has been used in feeding premature and new born babies. This has been true because of the established belief that these infants lacked the ferments requisite to the proper function of digestion. But late research shows that protein, carbohydrate and fat ferments are present in the gastro-intestinal tract very early in fetal life, pepsin having been found in the gastic mucosa even as early as the fourth fetal month. During the fifth and sixth months lipase and trypsin appear, while hydrochloric acid, rennin, secretin, erepsin, steapsin, maltase, diastase, ptyalin, invertin and saccharase have all been found at various later months. Lactase is one of the last ferments to occur and is therefore absent in some prematures, but makes its appearance at once when food is given. It can be said that all the necessary ferments are present at birth or as soon thereafter as food is administered, since food acts upon the ferments directly as a stimulant.

Lack of ferments then is not a cause of the difficulties and peculiarities of digestion in new-borns, but rather the cause is physical frailty. Prematures are naturally weaker than full term babies and in their feeding it is of highest importance not to overtax their physical capacity. Therefore it is essential to stress the principle of quality in solving the problems of feeding of prematures and new-borns.

In the solution of feeding problems of weak babies other than new-borns and of young children who are frail, the use of foods rich in nutritive

*From the Miller Hospital Clinic and the Pediatric Department of the University of Minnesota Medical School.

*Read before the Medical Forum, December 6, 1922.

value which could be given in small quantities is not a new thing. Two such foods are Keller's malt soup and thick cereal feeding. The former was valuable not only because of its composition but also because considerable nutrition could be given in small volume. Ingredients: one-third litre of whole milk, 600 grams of water, 100 grams malt extract, and 50 grams flour. The value of thick cereal feeding in pylorospasm and pyloric stenosis has long been firmly established, since in these conditions it is especially important that the retained food be of high caloric value. The many excellent results reported have shown the wisdom of this food even in spite of the fact that starch may cause severe disturbance in infants and young children.

Among the present day preparations receiving most attention are:

1. Thick cereal mixtures with the directions given by Sauer forming a basis for various formulas as used by Graves, Chapin, Mixsell and others. Sauer's directions: To 9 ounces skim milk and 12 ounces water add 6 tablespoons farina and 3 tablespoons Dextri-Maltose. Cook until thick.

2. The Dubo of Schick, whole milk with 17 per cent sugar.

3. The concentrated eiweiss of Finkelstein, prepared as the original eiweiss without the addition of water, plus 10 to 20 per cent carbohydrate.

4. The Czerny—Kleinschmidt butter-flour mixture. Ingredients: 7 grams butter, 5 grams sugar, 7 grams flour, 100 c.c. water. To be mixed with milk as indicated. For ease of preparation three times these amounts can be used. The butter is heated in a pan over a slow flame until bubbles appear and any odor of volatile fatty acid has disappeared. To this the flour is now carefully added and both ingredients again gently cooked with continuous stirring until they become a thin, smooth brown mixture,—to which is added the previously warmed water in which the sugar has been dissolved. The mixture is again brought to a boil, strained and then added to the desired amounts of milk.

5. The butter-flour whole milk and the butter-flour cereal of Moro. The butter-flour whole milk is composed of: 5 grams of butter, 3 grams of flour, 7 grams of sugar, and 100 grams of whole milk. This is prepared much the same as C.-K. butter-flour mixture except that the volatile fatty acids are not driven off by heat.

The butter-flour cereal is made with: 5 grams of butter, 7 grams of flour, 5 grams of sugar, and 100 grams of whole milk. This is boiled to a cereal and fed with a spoon.

In the discussion of the value of the concentrated feedings there is much of interest. That the thick cereal feedings are of decided value in pylorospasm most investigators agree. Since the work of Sauer many results have been published. That it has a quieting effect on a sensitive, irritable pylorus there is no doubt. It also prevents aerophagy, which is an important element in the production of habitual vomiting. Also due to its composition it cannot be vomited as easily as the thin mixtures. Preliminary gruel feedings often stop a mild vomiting in breast fed infants in a day or two. As a complemental feeding to the breast fed infants who do not thrive in the first six months it is excellent because it adds several carbohydrates to the diet in concentrated form without the giving of much fluid. Its use, however, is limited to those infants without a nutritional disturbance. Many pediatrists claim that breast fed infants take the gruel feedings earlier than those artificially fed because the breast milk furthers the digestion of the starches. In exudative diathesis of the edematous form it allows the giving of food without the additional amount of fluid. In constipation it adds bulk and may be very laxative if sufficient carbohydrate is added. In chronic bad feeders good results are published by Chapin, Mixsell and Graves. Chapin reports a series of twenty cases of marasmus treated with whole milk mixtures, with the addition of flour and sugar in various proportions, in which the results were for the most part good and an analysis of the stool showed an assimilation of carbohydrate almost comparable to that of normal children. Graves had good results with thick cereal mixtures made with farina and cream of wheat, which he prefers because of their property of expansion, making a thick mixture with the addition of a minimum amount of starch. He lays emphasis upon the examination of the stools. However, the presence of starch in the stools if symptoms of colic are absent is not of great importance. In normal nursing babies an insufficient supply of breast milk may be supplemented by the cereal feedings, and the infant who receives solid food early in life has a firmness and an excellent muscle tone that is lacking in babies kept too long on a milk diet only. The early feeding is also an

advantage in paving the way to the easy addition of other foods when the time comes.

Results with the Dubo feeding (whole milk and 17 per cent sugar) are published by Schick, Schick & Helmreich, von Groer, Heller, Davidson and Lust. Schick uses this preparation in the new-born ward and gives the infants food on the first day of life. His results are very good. The so-called physiological loss of weight is usually prevented and many gain from the first day. Vomiting is not frequent. The stool changes from a meconium to a food stool on the second day of life. As soon as the mother's milk can be obtained in sufficient amounts the substitute feedings are omitted. Heller, following the teaching of Schick, had good results in prematures; he found the food excellent due to the small amounts necessary to produce growth. Beginning the feedings a few hours after birth, he found that loss of weight could be prevented and that new-borns show the same tendency to rapid growth as the fetus if only enough food is given. However, he agrees with Von Reuss that they must have the ability of water retention. After the new-born period his best results are with Dubo as an additional feeding to human milk. With Dubo given alone his observations show three stages: marked thriving, weight at a standstill, and loss of weight. This was after weeks of continued feeding. Schick emphasizes that the tolerance of the child must be carefully watched if the feedings are to be given over long periods of time, and that as soon as any dyspeptic symptoms appear a lowering of the food to the minimum is necessary and then a gradual return to the optimum. Lust had excellent results with new-borns. He knows no artificial food that gives such excellent gain in weight, and finds it even better than breast milk. But he finds that the percentage of children who go over into dyspeptic conditions after its long continued use is not small. In combination with breast milk his results were good and dyspepsia was seldom seen. Von Groer had excellent results in the acute infectious diseases and dysentery, with Dubo plus the other concentrated foods.

In the Czerny-Kleinschmidt butter-flour mixture the authors attempted to make a food very similar to breast milk in composition. The fat and carbohydrate percentages are kept high and the protein is low. The carbohydrate is partly starch which is dextrinized by heat in the preparation. They lay stress upon its value as a food owing to: (1) the driving off of the volatile fatty acids; (2) an amount of flour equal to that of the fat; (3) chemical alteration of the flour; (4) the high carbohydrate and fat percentages; (5) the low protein. They advise the food for young and premature infants much below weight who have not been made to thrive on other foods, having a special value alternated with breast milk. They do not recommend it for infants with acute digestive disturbances, with or without fever.

Not all agree that much of its value lies in the fact that the volatile fatty acids are driven off by heat. Moro especially argues against this point, and in his butter-flour whole milk we have a preparation with the same ingredients as the Czerny-Kleinschmidt formula but in the making of which the volatile fatty acids are not driven from the butter. The relation of fat to carbohydrate is as 1:1.7, which is the same as that of human milk. The results reported by Moro and his followers are quite as good as with the C.-K. preparation.

As a standard artificial food for all cases no preparation can be discussed as such. High fat, high sugar, and high protein formulae have their limited uses, but none take the place of human milk in infant feeding. When we consider that many artificially fed infants thrive on very rich mixtures it becomes evident that the powers of assimilation vary within wide limits. This is as a rule for short periods of time only, for sooner or later a high percentage of these children show symptoms of dyspepsia. For good thriving a food must contain at least the minimum requirement of all elements necessary for growth. This was not true of the old third milk mixture and its use has practically been discontinued in all clinics. High fats and high proteins are usually well borne for long periods of time and the onset of dyspeptic symptoms is slow. With the high sugar feedings the onset of any disturbance is usually rapid and a return to the normal is usually accomplished in a few days by a low carbohydrate feeding. The stage of repair after an exceeded tolerance with fat is usually slower. In the using of the concentrated preparations it becomes necessary to raise the percentage of fat and sugar beyond what was formerly regarded as safe for infants and young children. We know now that these high percentages can be given if we maintain a fairly definite relationship of the elements. High fats can be given with perfect safety if we raise the carbohydrate. This

knowledge has been used by Czerny and Kleinschmidt in their butter-flour mixture, and by Moro in his butter-flour whole milk. Following another well founded principle that high carbohydrate can be given with a high protein percentage, Finkelstein made his concentrated eiweiss milk. The reason for the success of the Dubo of Schick is also partly due to the high protein content of cow's milk.

We must also consider growth with the concentrated foods. Until the last few years the amount of fluid required for the thriving new-born was thought to be that quantity taken in breast milk. The work of Schick and Helmreich and that of Heller has shown that growth is in every way normal with the fluid reduced one-half. With a triple concentrated food growth took place equally well. The urine, however, became only a few cubic centimeters in amount, dark in color, and often showed traces of sugar. The temperature at times was elevated. These symptoms disappeared with the giving of more fluid and this fact is often used as an argument against the limited giving of water. For healthy infants with a good appetite who will take large quantities there is no reason for this limitation. It is in the dystrophic cases that the more concentrated preparations have their place. It is the under-nourished, non-thriving infant who needs much food. This has long been recognized, but to administer this amount of food has been the problem because of the dilutions used. These cases often have a bad appetite and are prone to vomit. With an increase in the quantity of food the vomiting increases and the condition becomes worse. With the thick preparations many of these cases show rapid improvement. In a recent paper Finkelstein says that we must free our minds of the traditional view that infants in a state of dystrophy cannot take foods without high dilutions. He also points to the work of Schick with Dubo and says that sugar is not to be feared as a cause of fermentative diarrhea and toxic conditions as was formerly believed. However, the fact remains that these infants do remarkably well on preparations where much nutritional value can be given in small quantities. Extra fluid can be given between feedings if it is the hot time of the year and necessary for the comfort of the patient.

SUMMARY

Food which can be given in small volume is indicated in:

A. The well child:
   1. Those who need to be fattened.
   2. Those with lack of appetite.
      (*a*) New-borns and prematures.
      (*b*) Anorexia.
         (*aa*) Of nervous origin.
         (*bb*) Following an acute illness.
   3. In infants with intertrigo and maceration of the skin due to constant wetting of their clothes.
   4. In enuresis.

B. The sick child:
   1. With anorexia—the influenzas.
   2. Those with continued vomiting.
      (*a*) Nervous vomiting.
      (*b*) Pylorospasm.
      (*c*) From severe coughing.
         (*aa*) Pertussis.
         (*bb*) Pneumonia.
      (*d*) In acute infectious diseases.
   3. Those with difficulty of taking food.
      (*a*) Sore throat.
      (*b*) Dyspnea—pneumonia, croup, diphtheria.
      (*c*) Disturbances of cerebral function—typhoid and meningitis.
      (*d*) Tetanus.
   4. In diarrhea.
      (*a*) Typhoid.
      (*b*) Dysentery.

As contra-indications:
   1. The healthy child who needs volume to satisfy the pangs of hunger.
   2. Cases of acute vomiting and diarrhea of alimentary origin.
   3. When children cannot be under absolute control.
   4. Not to be used over long periods of time but as a therapeutic measure only.

BIBLIOGRAPHY

1. Moro: Monatschr. f. Kinderh., xviii, 1920.
2. Heller: Monatschr. f. Kinderh., xix, 1921.
3. Helmreich u. Schick: Zeitschr. f. Kinderh., xxx, 1921.
4. Davidsohn: Ztschr. f. Kinderh., xxxi, 1922.
5. Graves: Am. Jour. Med. Sci., clxiii, 576, April, 1922.
6. Chapin: Arch. Pediatrics, xxxviii, 418, 1921.
7. Sauer: Am. Jour. Diseases Child., xxii, 166, August, 1921.
8. Lust: Berl. klin. Wchnschr., 1922, i, 1603.
9. Von Groer: Ztschr. f. Kinderh., 30, 1921.
10. Czerny and Kleinschmidt: Jahrb. f. Kinderh., 87, 1918.
11. Griffith and Mitchell: New York Med. Jour., cxiv, No. 3, 137, 1921.
12. Hess: Premature and congenitally diseased infants.
13. Finkelstein: Jahreskurse fur Arztliche Fortbildung, Heft. vi, 1922.

PREGNANCY WITH COMPLICATIONS: FIBROIDS; ECLAMPSIA; CESAREAN SECTION; ACUTE GASTRECTASIA; THROMBOPHLEBITIS: CASE REPORT

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The patient, aged 32, presented herself at the end of the first trimester. Aside from the present illness, the history is without interest. She believed herself to be pregnant, having menstruated for the last time on October 18, 1921. Previous to that she had had menstrual flow of moderate amount occurring regularly every twenty-eight days, lasting for three days without pain. At the first examination she complained of slight frequency of urination, poor appetite, occasional nausea and headache. The physical examination disclosed a nodular mass in the pelvis interpreted as an enlarged uterus probably of pregnancy with hard fibroid tumors through its body. The patient was carefully observed each month and the diagnosis corroborated. The blood pressure, urine and general condition remained entirely normal throughout the first eight months. At this time she suffered from false labor pains and was sent to the hospital on the suspicion that delivery might take place. The family was instructed that abdominal cesarean section might be necessary because of the fibroid condition of the uterus. As the pains stopped, however, the patient returned home. When seen ten days later, the urine and blood pressure were normal.

On July 21, 1922, three weeks after the false labor, the patient noticed spots in front of the eyes and a short while later had a convulsion. The blood pressure at this time was 160/120. Abdominal cesarean section was performed and a living baby delivered. Following the operation, the blood pressure was 140/100. She was placed on morphin and chloral hydrate, and after a small temporary rise in blood pressure, the tension fell on the following morning to 136/88 and remained in that neighborhood. On the second postoperative day, she complained of great distress and 32 ounces of a dark brown fluid containing flocculent material was removed from the stomach. An attempt was made to insert a Rehfuss tube, but the patient could not swallow it; so the stomach was washed out through a stomach tube with water containing 2 grams of tannic acid. Five minims of pituitrin were given hypodermically every six hours. This resulted in a slight rise in blood pressure, but gave the patient much relief. At the same time enteroclysis was started. On the tenth postoperative day, the temperature was elevated and the cervix was dilated. The fever dropped in three days, but on the fourteenth postoperative day, the patient complained of pain in the left calf. Several small masses were distinctly felt near the surface, and the limb was raised and surrounded with ice caps. The thrombophlebitis was completely relieved by the sixteenth postoperative day and the patient left the hospital on the twenty-second day. Unfortunately, in spite of the care of capable pediatrists, the baby died on the fourteenth day.

The presence of fibroids in the uterus is a subject which requires careful consideration as to prenatal care and management of the delivery. The uterus on exposure in this case showed such a multiplicity of nodular growths that for this condition alone, cesarean section would likely have been necessary. In addition to the larger growths which could be palpated through the abdominal wall there were many (perhaps 200) smaller masses scattered over the entire surface and planted in the various muscular layers of the uterus which had not been palpated previous to operation.

Among other things, eclampsia has been attributed to infarct formation in the placenta with absorption of the autolyzed tissue. In this case, there was present on the surface of the placenta an area approximately three-fourths of an inch in diameter which showed degeneration, and might probably have been loosened at the time of the false labor pains, since previous to the actual appearance of the convulsions there was little indication of eclampsia. There is a difference of opinion as to the treatment of choice in eclampsia, though it may be said that operative treatment certainly offers a lower mortality to the infant and perhaps also to the mother over the expectant treatment. If operation is to be performed, it should be done not later than six hours after the first convulsion.

Acute gastrectasia is a not uncommon condition, though its presence is usually an indication of a weakened patient, since it most frequently occurs in patients in poor condition. Its mortality is variously estimated at from 50 to 75 per cent. The seriousness of this condition is easily offset by early recognition and prompt treatment. The stomach tube has long been recommended as the most effi-

cient remedy for gastrectasia. The action of the tannic acid which was utilized in this case serves to coagulate the blood and stop bleeding points in the gastric mucosa, in addition to acting as an astringent. Pituitrin acts as a stimulant to smooth muscle, thus serving to bring the stomach to its normal size and function. Repeated small doses extend this effect over a longer period of time.

The slight retention of lochia on the tenth day resulted from the insufficient dilatation of the cervix and secondary to this was the thrombophlebitis which occurred in the left leg. It is well to watch for retention in cases which have been delivered via cesarean section, since seldom has the cervix been dilated and the lochial flow may be slightly checked. The severity of the thrombophlebitis may be aggravated by failure to recognize the source and promptly eliminate it.

There were two indications for cesarean section in this case: first, the fibroids in the uterus, and, second, the eclampsia. Conditions tending to infection contraindicate this proceeding. Vaginal examination had been done early in the onset of gestation and strict hygiene had been observed, so that the possibility of infection was so slight as to justify immediate operation. It might be questioned why the Porro cesarean section was not done, but the patient's condition would not warrant it.

TERSUL-HILLER NOT ACCEPTED FOR N. N. R.

The Council on Pharmacy and Chemistry reports that Tersul-Hiller (Robert Wollheim, distributor) is a German preparation of silicon and calcium proposed as "An Adjuvant to the Treatment of Tuberculosis, Rachitis, etc." Physicians are told that Tersul-Hiller is "composed essentially of coincidently water-soluble silica and water-soluble calcium salts as shown by the fololwing approximate percentage formula: Silica (new process) 4½ gr., calcium lactate 7½ gr., calcium hypophosphate ¾ gr., calcium phosphate 7½ gr., calcium fluorid 1½ gr., calcium carbonate 60 gr., magnesium phosphate 3 gr., milk sugar 15 gr." From this formula it would appear that Tersul-Hiller is a mixture containing a silica compound of undeclared composition and a mixture of soluble and insoluble calcium compounds —chiefly calcium carbonate. The Council found Tersul-Hiller inadmissible to New and New-official Remedies because (1) there is no acceptable evidence for the therapeutic use of silica preparations; (2) the identity of the silica compound is not declared; (3) the use of the mixture is irrational, and (4) the claims for its therapeutic effects are unwarranted.—*Jour. A. M. A., May 24, 1924, p. 1712.*

A CONSIDERATION OF POSTOPERATIVE COMPLICATIONS FOLLOWING THYROIDECTOMY

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A review of the literature on surgery of the thyroid gland disclosed very little data on operative complications and treatment following thyroidectomy, practically all articles dealing with surgery of the thyroid being on preoperative treatment, actual technic, or end-results. In view of the fact that the recognition of such complications and treatment have such a definite bearing not only on the immediate, but on the ultimate results, I reviewed the case histories in the Mayo Clinic with regard to the postoperative complications of about two thousand patients on whom thyroidectomy had been performed for exophthalmic goiter and adenomatous goiter, and personally observed approximately two thousand more.

The purely surgical complications following thyroidectomy fall naturally into two definite groups: (1) those due to a pathologic condition present at the time of the operation, or to a coincident surgical complication developing after operation, and considered unavoidable; and (2) those that are accidental and dependent on the technic of the operation and the personal skill of the operator; these may be considered avoidable in the majority of instances.

Group 1. — Among the unavoidable complications, "collapsed trachea" has been held responsible in many cases in the past. This condition is a definite clinical entity usually occurring in cases of large or hard, nodular goiters. It[4] is caused by softening of the tracheal rings, the result of pressure. The trachea is held patent by the fascial attachments to the enlarged gland. Thyroidectomy removes the supporting structure, and if there is severe erosion of the tracheal rings, collapse may follow. The inciting cause of the collapse may be rotation of the trachea during operation, pressure from edema, or hemorrhage in the surrounding tissues, or the inspiratory effort, causing the walls to fall together, and act as an obstructive valve. These complications may occur during operation or within a few hours after. Some years ago, difficulty in breathing following such operations was

ascribed to collapse of the trachea, the responsibility of which was not assumed by the operator. In recent years, however, it has been learned that such dyspnea rarely is due to collapse of the trachea, but usually to some other condition. In none of the cases in this series which came to necropsy, could the cause of death be ascribed to collapse of the trachea. When it does occur, immediate tracheotomy, to avoid death by asphyxiation, is necessary. The trachea must be supported either by a tracheotomy tube, sutures, or packing, until sufficient adhesions have formed to hold it patent.

Tracheitis and laryngitis, with the edema of the trachea and larynx, are present to a certain degree in a large percentage of cases following thyroidectomy. This is the result of manipulation, of mechanical injury to the trachea because of its immediate proximity to the field of operation, and of alteration of the blood supply in this area. The closer and more extensive the association between the thyroid and the trachea, the greater will be the reaction. In most instances, the reaction will be mild and transient, manifesting itself by an increased amount of viscid mucous sputum, and a thick, hoarse voice; it may, however, be much more serious. The onset of the edema in this type of case is usually within two to four hours after operation. The patient gradually becomes hoarse, and breathes with increasing difficulty. Injury to the recurrent laryngeal nerve may or may not accompany this condition. Laryngoscopic examination will reveal edema of the larynx and the trachea and vocal cords; in some cases the air passage will be narrowed markedly. This condition rarely, if ever, progresses to the point of operative interference unless accompanied by injury to the recurrent laryngeal nerve. The constant inhalation of steam with compound tincture of benzoin affords the greatest relief to these patients. The patient should be entirely inclosed in a tent, so that he will constantly breath an atmosphere saturated with steam. In twenty-four hours the patient will usually be markedly improved.

Occasionally coincident surgical conditions may be included under the heading of unavoidable complications, although they are naturally rare. Two cases were noted in which acute appendicitis developed within forty-eight hours after a thyroidectomy. The patients were operated on and convalesced uneventfully, although there must have been a very definite increase in the operative risk. In a case of acute cholecystitis with stones following thyroidectomy for exophthalmic goiter, the patient was treated medically and carried through the postoperative stage. Another patient developed definite signs of intestinal obstruction a few days after the original operation, but symptoms subsided before operative interference was necessary. Menstrual complications are sometimes distressing, and, though not dangerous, may definitely prolong convalescence. One patient had a flare-up of an old salpingitis several days after operation. The condition was treated for about three weeks, when operation was performed.

Group 2.—Among the accidental complications that are dependent on the technic and skill of the operator, paralysis of the vocal cords, due to injury of the recurrent laryngeal nerve, is by far the most serious. From the standpoint of complications and the relation to operative risk, such injuries are of two types: transient paralysis,[11] the result of pressure or manipulation of the recurrent laryngeal nerve, and permanent paralysis,[3] the result of clamping, cutting, or ligating the recurrent laryngeal nerve. The first type, which has little effect on the convalescence, may be manifested by partial or complete abductor paralysis of the vocal cord. It is sometimes difficult to determine whether this is due to local edema, or to partial or temporary nerve injury. The paralysis usually clears up in from one to three weeks. If the recurrent laryngeal nerve is cut, clamped, or ligated, there is complete paralysis of the corresponding vocal cord, which is usually permanent. In a few cases in which the nerve is only torn or crushed, regeneration may occur, and the vocal cord return to normal after several months. The paralysis may be either unilateral or bilateral; it has a definite bearing on the immediate convalescence, and on the future well-being and happiness of the patient.

Injury to one nerve may result in definite hoarseness, or there may be no perceptible change in the voice. There may or may not be signs of obstruction to breathing. If dyspnea is present, it is usually complicated by local edema. Regardless of whether or not this is the case, the convalescence of the patient is usually definitely protracted. Examination of the vocal cords may reveal the cord on the side of the injured nerve fixed in the abductor or in the cadaveric position, and without movement on attempted phonation. However, with one normal cord there is not sufficient obstruction to

affect materially the every-day life of the patient after recovering from the operation. The voice usually approaches the normal as the other vocal cord hypertrophies in accommodation. There is little doubt, however, that in the presence of unilateral vocal cord paralysis,[8] there is a definite increase in the mortality in event of complications such as a crisis, pneumonia, or acute infectious disease.

Bilateral vocal cord paralysis[5] due to injury to both recurrent laryngeal nerves is a serious complication. The immediate effect on the voice is not always the same. The patient may still be able to talk, but with a peculiar, characteristic, brassy type of voice. When once heard, this type of voice will always associate itself with paralysis of the vocal cords. The patients may or may not have immediate partial obstruction to breathing. If it does not occur at once, it usually develops to some degree within twenty-four hours. Frequently dyspnea, and at times stridor, are the only symptoms. Laryngoscopic[7] examination of the vocal cords of this type shows both cords fixed in the abductor or median line position with a very narrow slit between them for the passage of air. Patients must be watched carefully, by an experienced nurse, as their surgical risk is greatly increased. Any transient, acute infection might be sufficient to cause death. A tracheotomy tray should be kept in the room, and all in readiness for tracheotomy at a moment's notice. Steam with compound tincture of benzoin should be in constant use with the patient in a tent.

Another type of bilateral cord paralysis is not as distressing, or as serious from an immediate standpoint. The patient has complete loss of voice and talks in a loud falsetto whisper. There is no obstruction to breathing. Drinking water causes a very distressing choking. This is followed usually by coughing and straining. Laryngoscopic examination reveals fixation of the vocal cords in the intermediate or cadaveric posittion. There is no motion of the cords when talking is attempted. Although there is no obstruction to breathing, the possibility always exists that it may develop suddenly. Here also it is wise to have a tracheotomy tray in the room in preparation for emergency. After a prolonged convalescence the patient usually goes home feeling fairly well except for loss of voice and a tendency to dyspnea on slight exertion. Examination of the vocal cords after a few months will show them to be contracted to the abductor position. Patients gradually learn to talk in a weak, cracked voice, but are unable to lead a normal existence because of the ever-present dyspnea caused by slight exertion. They may be semi-invalids for years, always living in fear of exerting themselves and of the possibility of a complete obstruction to breathing. Sooner or later attempts must be made to alleviate this condition.

In connection with injuries to the recurrent laryngeal nerve it may be well to mention hysterical aphonia, which should be distinguished from injury to the nerve. The onset may follow operation directly, or it may not arise for several days. In most instances, the voice returns spontaneously. Laryngoscopic examination will at once differentiate this from true vocal cord paralysis.

Hemorrhage following thyroidectomy is another very important accidental complication. There are two distinct types: one occurring within the first forty-eight hours; the other delayed, manifesting itself from ten to twenty days after operation. The first is by far the most serious. It has a very definite effect on the convalescence of the patient, and unless apprehended and arrested early, may be the direct cause of death. If there is an unusual quantity of blood in the dressings, requiring changing more than once or twice in twenty-four hours, the patient should be watched closely for hemorrhage. Seeing the wound with the dressing off will usually determine whether or not the drainage is excessive. If the flow of blood becomes a steady dropping or a small stream, the wound should be opened and the hemorrhage stopped. The bleeding usually comes from a branch of the inferior thyroid artery, from one of the lateral veins, or from a general oozing of the tissues. Undoubtedly the slipping of a ligature during a coughing or vomiting spell is directly responsible for it in many cases.

Hemorrhage[9] occurring early following thyroidectomy may be accompanied by clot formation. It is much more common and dangerous than the former type. It is caused by slipping ligatures, small vessels overlooked at operation, vessels crushed and starting to bleed during a coughing or vomiting spell, or a general capillary oozing. There are several characteristic symptoms which should be recognized early. The first is practically always mentioned by the patient himself. If he complains that the dressing is getting tight, the wound should be examined. If the bleeding is not apprehended

at this stage, it is usually progressive. The symptoms, as the clot grows larger, occur in the following order: difficulty in swallowing, feeling of a lump in the throat, difficulty in breathing due to pressure on the trachea, and finally complete loss of voice. These symptoms are undoubtedly due to a combination of pressure on the recurrent laryngeal nerve, pressure on the trachea, and the resultant traumatic edema of the surrounding structures. If the symptoms are progressive and the condition not recognized and relieved, the patient will go on to asphyxiation and death. Prompt opening of the wound, evacuation of the clot, and ligating of any bleeding points, or, if none can be found, packing of the wound, will relieve all symptoms with very little increase in the mortality rate.

Late postoperative hemorrhages occur but rarely, and then only in the presence of a particularly virulent type of wound infection. One case was noted in which the hemorrhage occurred on the fourteenth day. The patient had an infected wound and had been having hot dressings. The character of the drainage had become serous, and the patient was dismissed from the hospital. He was brought back on the fourteenth day, bleeding profusely from the wound. Operation disclosed the offending vessel to be a large branch of the inferior thyroid artery which had been eroded by the infection. The vessel was ligated, and the patient recovered uneventfully.

Edema[6] and ecchymosis of the skin flap do not form a dangerous complication, but may definitely prolong convalescence. The edema itself rarely affects more than the flap. The ecchymosis, however, may be widespread, and involve all the wound area and a greater portion of the chest wall; thus the wound drains longer, and in case of contamination a fertile field for infection is afforded. Sterile hot dressings should be applied every two to three hours for several days.

Infection of the wound is not common, but when it does occur, it assumes a variety of forms and may result in protracted and long drawn out convalescence. Practically all wounds following thyroidectomy were drained. In a few cases, gauze packs were used, and in a few others, wounds were packed with gauze and left open. Following the sero-sanguineous drainage of the first few days, the character of the drainage changes to serous. Cultures of the wounds in this type, taken from the tenth to the fifteenth day, showed either streptococcus or staphylococcus in large numbers. In most instances patients recovered uneventfully without general symptoms of infection. From the standpoint of effect on the convalescence, such wounds cannot truly be considered infected. In a second type there was a sero-purulent drainage. In these, also, cultures were positive, but they usually cleared up after a moderately extended period of drainage. In a third type the infection was more severe, with a frankly purulent drainage. Cultures in most of these cases showed *streptococcus hemolyticus*. Such wounds require sterile hot dressings applied every two to three hours for a considerable period. In the fourth type the wounds drained very little or none, at first. The temperature varied from 100° to 103°, and there was a general feeling of intoxication. The wound was indurated, and somewhat swollen, and the flap reddened. Hot dressings usually produced a purulent drainage within a few days. *Streptococcus hemolyticus* was present. The fifth type is very rare and apparently is caused by an extremely virulent type of organism. The drainage is purulent and profuse with marked sloughing of the flap, causing an ugly scar when the wound heals. Because of the preponderance of operations for goiter in women, the appearance of the scar becomes important. A good scar, in the opinion of the patient, may mean the difference between a very successful operation and a mediocre job. Ugly scars are practically always the result of extensive infection. It[1] is a simple matter, after the wound has healed, to excise this scar tissue and approximate the skin and muscles. Keloid formation is not common, but when present it can usually be controlled by mild doses of *x*-ray. The skin may sometimes be attached to the trachea at the point of drainage; swallowing causes an elevation of the skin at this point. If this still persists two or three months after operation, daily massage of the neck with cocoa butter will often cause stretching of the scar tissue so that it will not be noticeable. If this does not improve the condition, excision of the scar and approximation of the skin and muscles will remove the deformity.

Air embolism occurs infrequently, and is an operative, rather than a post-operative complication, the result of tearing a large vein, or failure to ligate properly the lateral veins. Three cases occurring on the operating table have been noted. If a large vein is open, air may be sucked in during inspiration. The symptoms are those of cerebral

anemia accompanied by a cessation of respirations, and if correctly interpreted early, prompt lowering of the head of the table and ligating the vein will always bring these patients through. The complications occurred in cases of large adenomatous goiter with enormously distended and tortuous lateral veins. All the patients recovered.

Tetany[2] as a postoperative complication is now far less common than in the past. It is the result of removal of part or all of the parathyroid bodies, or of interference with their blood and nerve supply. Since the days of complete lobectomy, it is very unusual to remove the parathyroid glands during a thyroidectomy. However, since the adoption of resection rather than complete removal of the thyroid gland, most of the cases are due to interference with the blood or nerve supply. There are two very definite types of postoperative parathyroid tetany, transient and permanent. In the transient type, which is undoubtedly due to interference in the blood or nerve supply to the gland, a heavy milk diet, and calcium lactate by mouth, will usually cure the condition in a few days. For the attack itself, 5 c.c. of a 10 per cent solution of calcium chlorid intravenously will produce immediate relief. In the permanent type treatment with calcium chlorid and parathyroid extract will hold off attacks, but cessation of the treatment is soon followed by their recurrence. In this type there is definite and permanent injury to the parathyroid glands, or to their blood supply. There were no cases of permanent tetany in this series.

Myxedema is an unusual sequela of thyroidectomy. It is caused by the removal of too much gland tissue, infection of the wound with resultant sloughing and destruction of gland tissue, or by the presence of a thyroiditis at the time of operation. The medical treatment of this condition with thyroid extract and thyroxin will re-establish a normal rate.

The incidence of purely medical complications is mentioned chiefly to emphasize the additional complications which must be dealt with. The accompanying symptoms and their treatment will not be discussed in detail.

The crisis of exophthalmic goiter, or the onset of acute hyperthyroidism following operation, is not nearly so common as formerly. When it does occur, the usual treatment of heavy doses of Lugol's solution,[10] digitalis, icebags, forcing fluids by rectum and subcutaneously, with nothing by mouth, and the liberal use of morphin to combat the restlessness, are the chief forms of treatment.

Acute dilatation of the heart is rarely found except in crisis. Digifolin hypodermically or intravenously seems to be the most desirable form of digitalis. Auricular fibrillation is the usual rather than the unusual condition in the severely toxic cases. Auricular flutter is much more infrequent. The usual treatment by morphin, ice cap to precordium, and digitalis is used.

Pneumonia as a complication is rare, since the use of local and combined anesthesia. It is practically always of the bronchial type and rarely fatal. Alone it does not constitute a very markedly increased risk. In the presence of injuries to the recurrent laryngeal nerve and subsequent vocal cord paralysis, however, the risk immediately goes much higher. The complications of pneumonia, such as dry pleurisy, pleural effusion, or empyema, must be dealt with as such. There is a definite increase in the mortality if these occur early.

Acute follicular tonsillitis sometimes occurs. One case in particular is mentioned because of the fact that the patient, on being questioned, insisted that his throat was not sore. His throat therefore was not examined in the search for the cause of his toxic condition, and the condition was overlooked for twenty-four hours. On examination, the throat was found to be acutely inflamed. The usual hot saline irrigations and steam inhalations suffice to take care of this type of complication.

Most types of acute infectious diseases have been seen at one time or another. Nothing more can be said than that isolation of the case and treatment of the condition must be instituted. The risk is, of course, definitely increased.

Phlebitis following thyroidectomy is rare, and with it there are usually coexistent varicosities. One case was noted. Treatment consisted in rest, elevation, and heat to the limb. Recovery was uneventful.

SUMMARY

The present day interpretation of surgical complications is radically different from that of ten to fifteen years ago, especially with regard to complications causing interference with respiration. We know that practically all of these are due to injury to the recurrent laryngeal nerve, or to pressure on the trachea from clot formation in hemorrhage. Many surgical complications do not markedly

affect the mortality rate, since they are promptly recognized and treated. The most serious of the complications from the standpoint of mortality and the future welfare of the patient is injury to the recurrent laryngeal nerve. The accurate checking by laryngoscopic examination is the only method of determining the degree and type of involvement of the vocal cord; on this, the prognosis depends.

BIBLIOGRAPHY

1. Crile, G. W., and associates: The thyroid gland. Philadelphia and London, W. B. Saunders and Co., 1922, 288 pp.
2. Crotti, André: Thyroid and thymus. Philadelphia and New York, Lea and Febiger, 1922, 774 pp.
3. Greenfield, W. J.: Personal communication, 1923.
4. Hertzler, A. E.: Diseases of the thyroid gland. St. Louis, C. V. Mosby Co., 1922, 245 pp.
5. Judd, E. S.: Laryngeal function in thyroid cases. Ann. Surg., 1921, lxxiii, 321-327.
6. Lauddie, A. K.: An unusual post-operative phenomenon in thyroidectomy. Indian Med. Gaz., 1922, lvii, 95-96.
7. Matthews, J.: Observations on the vocal cords in one thousand cases of goiter. Jour. Am. Med. Assn., 1910, lv, 826-827.
8. Pemberton, J. deJ.: Mortality in surgery of exophthalmic goiter. Surg., Gynec. and Obst., 1923, xxxvi, 458-462.
9. Pemberton, J. deJ.: Personal communication, 1923.
10. Plummer, H. S., and Boothby, W. M.: The value of iodin in exophthalmic goiter. Jour. Iowa State Med. Soc., 1924, xiv, 66-73.
11. Sistrunk, W. E.: Personal communication, 1923.

A little more than fifteen years ago, on Memorial Day, a wreck on a trolley line at Elyria, Ohio, caused the death of sixteen passengers and sent half a hundred others to a small local hospital in an old residence. The lack of adequate facilities caused the death of a number, among them Homer Allen, a high school boy, son of Edgar F. Allen, a business man of Cleveland, but a resident of Elyria. The utter inadequacy of the hospital to meet any emergency, its incompleteness even for ordinary conditions, greatly impressed Mr. Allen. It did more; it caused him to resolve to give up his million-dollar business and devote his energies, time and money to ameliorating suffering. He headed an organization to provide Elyria with a hospital that would meet all present and future needs. Large, fine grounds were purchased, and a modern hospital, costing $100,000, was built. He gave liberally himself, besides proving himself a splendid solicitor. The town was rapidly growing from a beautiful village to a manufacturing city, and five additional units to the hospital have been built. Today the outlay represents $1,000,000. Mr. Allen assumed the general management without salary, and when the monthly balance showed a deficit he made it up from his own pocket.

About ten years ago a local physician remarked casually that something ought to be done for the crippled children of the city. Mr. Allen thereupon investigated and found that the United States possessed only three crippled children's hospitals, one at Canton, Mass.; one at Haverstown, N. Y., and a third at St. Paul, Minn. He learned all he could of the work being done, but he doubted whether there were enough cripples in Elyria or in the county to justify the erection of a hospital exclusively for them. Investigation, however, soon brought to light twenty young cripples in the city and more than 200 in the county. Mr. Allen's interest grew, and he employed at his own expense a capable woman to survey several nearby cities. He found there was one cripple to every 500 of the population. He has since found through surveys in many large cities that there is a crippled child for every 400 of the population. This would indicate no less than 325,000 in the whole country. Soon after the county survey was made Mr. Allen presented his findings to the Hospital Board of Elyria and urged the building of a crippled children's hospital, to be placed upon the ample grounds of Memorial Hospital. The request was a distinct surprise and it was doubted if the community could at that time add this unit, but so great was the confidence in Mr. Allen that he was told to proceed with the scheme, if he could raise the money. A single donor gave $25,000 as a memorial to her husband, W. N. Gates. In this way the Gates Children's Hospital was founded.

The Edgar Allen idea of local rather than large State or regional hospitals is based upon the economy of the small hospital or ward. They should be in connection with large hospitals to secure the advantage of trained nurses, good medical attendance, nearness to the children's homes and the avoidance of red tape. The academic and industrial education with a vocational turn adapted to the child's possibilities after leaving the hospital has developed wonderfully in some of the cities. It is found advisable to establish day schools for their special benefit in the larger cities. Cleveland has 138 such children in day schools and is preparing for 250 more now on the waiting list. Cincinnati has 120, Toledo 125, Akron 37, with 90 on the waiting list, and so on through a long list of smaller cities. What a contrast this picture presents to that of European cities, where begging cripples line the streets! Not all children can be discharged from the hospitals fully cured or normal, but none is accepted unless he can be benefited. To make the work more effective Mr. Allen is now stressing the need of convalescent homes to relieve the hospitals after operations and to provide better educational facilities and more homelike surroundings.—*Current History, April, 1924.*

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS
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Name	*Medical College*	*Address*
Arthur, Francis Harding	U. of Minn., M. B., 1923	St. Barnabas Hosp., Mpls.
Carlson, Harold Wesley	U. of Minn., M. B., 1922; U. of Minn., M. D., 1923	Henry Ford Hosp., Detroit, Mich.
Colberg, Ernest Johnson	U. of Minn., M. B. & M. D., 1924	St. Peter, Minn.
Gupte, Vasant S.	U. of Minn., M. B., 1923	St. Mary's Hosp., Duluth
Jacoby, Lionel Arnold	U. of Minn., M. B., 1923	1917 Emerson Ave. S., Mpls.
Kitchen, Hubert Daniel	U. of Manitoba, M. D., 1921	Rochester, Minn.
Lapp, Victor Roy	McGill, M. D., 1921	Rochester, Minn.
Noble, Thos. Paterson	U. of Edinburgh, M. D., 1913	Rochester, Minn.
Rasmussen, R. Carl	U. of Minn., M. B., 1922	St. Mary's Hosp., Duluth
Settelen, Max Ernst	Basel, Switzerland, 1921	Rochester, Minn.
Travis, Walter T.	Cen. U. Ky., M. D., 1907	Ely, Minn.
Wheeler, Dan. Wilbur	Rush, M. D., 1923	Nopeming, Minn.

By Reciprocity

Blackstone, Geo. R.	N. W., M. D., 1900	Eveleth, Minn.
Campbell, John Wm.	Rush, M. D., 1897	Fargo, N. D.
Counseller, Virgil Sheetz	Rush, M. D., 1920	Rochester, Minn.
Dies, John Livengood	U. of Pa., M. D., 1918	Rochester, Minn.
Griffith, Guy Evertt	N. W., M. D., 1920	709 Duff Ave., Ames, Ia.
Griswold, Lincoln Bon	Loyola, M. D., 1923	60 S. Lincoln Ave., Aurora, Ill.
Hargis, Estes Henry	U. of Pa., M. D., 1921	Rochester, Minn.
Harrington, Ethel R.	Rush, M. D., 1917	Rochester, Minn.
Killins, Wendell Allensworth	U. of Neb., M. D., 1921	Rochester, Minn.
Merrill, Elmer Forrest	U. of Mich., M. D., 1920	Rochester, Minn.
Monnich, Walter Arthur	Baltimore Med., M. D., 1910	108 Buckingham Apt., Mpls., Minn.
Myers, Fred Earl	U. of Pa., M. D., 1921	Ely, Minn.
Parker, Stephen Thos.	Creighton, M. D., 1921	Rochester, Minn.
Parson, E. Lillian Bendeke	U. of Ill., M. D., 1923	Elbow Lake, Minn.
Randall, Laurence Merrill	U. of Ia., M. D., 1921	Rochester, Minn.
Sansing, Campbell	Tulane, M. D., 1895	427 6th St. S. E., Mpls., Minn.
Simon, Harold Ewart	U. of Pittsburgh, M. D., 1922	Rochester, Minn.
Swanson, John Albert	U. of Louisville, M. D., 1921	Lowry Bldg., St. Paul
Sweetser, Horatio B., Jr.	Harvard, M. D., 1921	2509 Pillsbury Ave., Mpls., Minn.
Underhill, Marshall Scott	N. W., M. D., 1921	Rochester, Minn.
Veirs, Ruby Jayne Smith	U. of Louisville, M. D., 1920	1541 Lincoln Ave., St. Paul, Minn.
Wheeler, Roy McMillan	N. W., M. D., 1900	3980 Lake Park Ave., Chicago, Ill.

National Board Credentials

Henderson, Earl Fletcher	U. of Pa., M. D., 1918	Rochester, Minn.
Hufford, Alvin Ray	Loyola, M. D., 1923	Rochester, Minn.

MINNESOTA MEDICINE

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EDITORIAL

Homeopathy

Chapters in history come and go and often the close of the chapter is only realized in retrospect. The chapter in medicine which deals with homeopathy is from all appearances rapidly drawing to a close.

Ask the recent medical graduate what he knows about homeopathy and in all probability all he can tell you is that one of the former schools of medicine prescribed on the theory that like cures like and that drug potency increases with dilution; that neither of these theories work out in practice and that's that. He never heard of Hahnemann's third theory of the "psora" or miasm, which was supposed to manifest itself in some form of skin disturbance. As a matter of fact this third theory never attained much credence among his most ardent followers.

The German physician, Hahnemann, first propounded his theories at the close of the eighteenth century. It was then known that quinine was a specific for malaria and the thought occurred to Hahnemann that quinine administered to the normal human being might produce a reaction similar to malaria. He tried it on himself. It did. Ergo —*similia similibus curantur.*

Hahnemann's theories gradually spread all over Europe in spite of temporary adverse legislation in Austria and later in England. In no country did these novel ideas find such fertile soil as in our own. Towards the end of the last century homeopathy was flourishing and at its close there were twenty-one homeopathic medical schools in operation in America. Few of the present generation can realize the foothold homeopathy gained among the profession in this country nor the heated debates it caused in our ranks.

Today, just two homeopathic medical schools exist in this country and last year just forty-nine physicians of the school were graduated. Homeopathic physicians who wish to be known as such are comparatively few in number. Homeopathy has come but is now obsolete.*

What is the explanation of the fall of homeopathy? The main reason lies in the fact that in the light of modern scientific medical progress the theories of homeopathy do not hold water. Quinine destroys the *plasmodium malariae* in the blood and is not an instance of like curing like.

The power of publicity in remedying conditions is nowhere better illustrated than in its effect on homeopathic medical schools. In 1901 the American Medical Association began publishing a yearly list of medical schools in the country and their ratings. Since then the number of homeopathic medical schools has rapidly decreased from twenty-one to two. So many of the homeopathic schools had low ratings that together with the falling off in patronage they became discouraged and quit. Many a homeopathic physician realizing the true state of affairs sent his son to a regular school.

Homeopathy has made its contribution to medical progress. If it accomplished nothing else, it counteracted the practice formerly in vogue of administering large doses of drugs. Patients in many instances did as well or better on the almost negligible medication of this school. The present day tendency away from useless medication is doubtless the result of the influence exerted by the school of homeopathy.

*In the June issue of The American Mercury, Morris Fishbein presents the subject of "The Rise and Fall of Homeopathy" in an unbiased and very illuminating manner.

"Dr." Henry Ford

The feeling of criticism of the methods pursued by the management of the Ford Hospital on the part of the medical profession of Detroit attained some publicity when the Detroit Academy of Surgery declined the invitation recently to hold a meeting at the hospital.

As explained in the local medical society bulletin, "The medical profession objects to having Henry Ford, a man of unlimited wealth and power, a man who controls a publication, a man of strong prejudices, practice medicine by proxy."

The story goes that some years back Mr. Ford received what he considered an exorbitant bill for medical services rendered a member of his family. This experience is supposed to have been the inspiration for the establishment of his hospital on a one-price basis. The established rates are based on cost of hospital operation and cover professional services. Millionaire and day laborer presumably pay the same rate.

Report has it that under the direction of its first superintendent, who was well trained along the old lines of hospital management, the institution did not make expenses. The superintendent was dismissed and under the personal inspiration of Mr. Ford's private secretary, modern commercial methods were adopted. Publicity was given to the hospital's possession of a stethoscope, microtome, etc., and the staff of Johns Hopkins graduates only was advertised. Result—the hospital grew by leaps and bounds.

The reply of the surgeon-in-chief, Dr. Roy D. McClure, in a dignified way called attention to the phenomenal growth of the institution which, in his opinion, disproves the accusation of factory methods, and claimed the right to existence of a uniform fee scale.

The gist of the controversy is contained in two questions: first, the propriety of a hospital so closely connected with the profesional staff, advertising to the public; second, shall there be a uniform or flexible fee scale?

Most hospitals advertise directly or indirectly and many a physician benefits thereby. It were better if all hospitals adhered to the same policies. When, however, as in the Ford hospital, the physician is paid by the hospital, advertising on the part of the hospital amounts to the same as professional advertising.

Mr. Ford's quixotic attack on the flexible fee scale generally practiced by the profession is interesting. Only a man of wealth and strong conviction could so disturb the profession. Most hospitals have endowments and at that have difficulty in making expenses. It is not likely that any one hospital in a locality could adopt the Ford hospital system and survive. It would be a question of all or none. Such a system would doubtless be satisfactory to the profession from a financial standpoint, but would throw a greater patronage on free municipal institutions.

It is not generally realized how much medical attention is given voluntarily for little or no remuneration by hospitals and the medical profession. The relatively few large fees attract attention, only serve to commercialize medicine and often are unjustifiable. Sometimes they may act as a boomerang as in the case of Mr. Ford.

Fancy fees only serve to detract from the idea of service which we as a profession must firmly adhere to. The physician who renders service, as a rule, has no great difficulty in the way of financial compensation.

COMMUNICATIONS

435 Hamm Building
St. Paul, Minnesota
May 28, 1924

TO THE EDITOR:

I have just received a letter from Paris from my neurological confrère, Dr. Walton M. Kraus, of New York, but at present in France, calling my attention to Netter's and Urbain's recent work on the "Further Investigations of the Deviation of the Complement in Herpes Zoster."** These investigations indicate the identity of the causative agents of herpes zoster and chicken pox. Dr. Kraus states that from the clinical viewpoint it has been reviewed in America by McEwen (Arch. Derm. & Syph., 1920); Kraus (New York Medical Journal, 1921); Riggs (Minnesota Medicine, 1922), and that these authors believe that these two diseases are due to the same or very closely related causes.

Netter and Urbain have demonstrated (Comptes Rendus, Jan. 26) an antigen obtainable from the sera and crusts of cases of herpes zoster (not labialis, which shows none of the reactions to be described), which fixes antibody of cases of herpes zoster. Further they demonstrated that this antigen has the same action upon blood serum from cases of chicken pox and that antigen obtained from chicken pox crusts also fixed both chicken pox and herpes zoster antibody. They confirmed this on thirteen additional cases of zoster. They conclude from this that the crusts or scabs

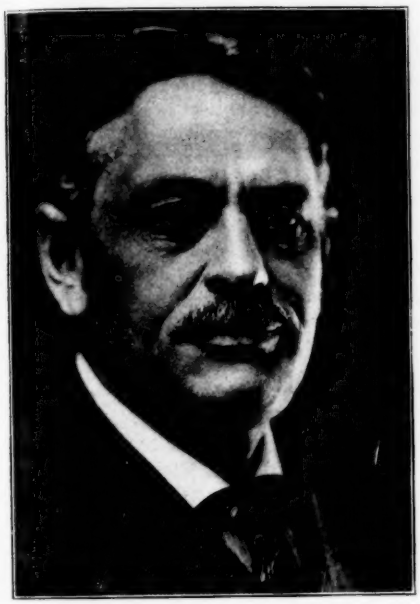
**Nouvelles Recherches sur la Deviation du Complement dans le Zona. Arnold Netter and Achille Urbain (Comptes Rendus. Soc. de Biol., Vol. 90, No. 7, p. 461 (1924).

of zoster contain an antigen capable of deviating the complement of the blood serum of both chicken pox and herpes zoster and that the converse obtains as well. Dr. Kraus believes that this bacteriological evidence added to the clinical evidence already accumulated emphasizes the probable identity of the causes of chicken pox and herpes zoster.

Very sincerely yours,

C. EUGENE RIGGS.

OBITUARY



DR. HALDOR SNEVE

Dr. Haldor Sneve, nationally known as a specialist in nervous and mental diseases and a resident of Saint Paul since 1899, died Sunday in San Diego, Calif., where he went a year ago in an effort to regain his health. June 15 1924.

Dr. Sneve formerly was president of the Ramsey County Medical Society, the Minnesota Academy of Medicine and the Minnesota State Medical Association, and in addition to holding numerous other offices in medical organizations during his career, he was president of the Town and Country Club, Saint Paul, at the time he went to California. Golf was his hobby.

He is survived by his widow, who made the journey West a year ago and was with him at the time of death.

Dr. Sneve came to Saint Paul in 1899 from Minneapolis, to become chief surgeon of the Chicago Great Western Railroad, a post which he held for thirteen years. He was a lecturer on mechanotherapy at the University of Minnesota from 1896 to 1899 and clinical professor of mental and nervous diseases from 1911 to 1914.

He was born in Albert Lea, Minn., October 27, 1865, and attended public school there. He was graduated from the Medical College of Ohio in 1887 and started his professional career as assistant surgeon in the National Military home at Dayton, Ohio, the same year.

From 1888 to 1890 Dr. Sneve was assistant superintendent of the Dayton, Ohio, hospital for the insane. He was the author of numerous medical papers and a member of many medical and scientific societies. He married Miss Katherine Stickney in 1897.

Dr. Sneve was a member of the Minnesota National guard for three years and served on the draft board during the World War.

REPORTS AND ANNOUNCEMENTS OF SOCIETIES

IMPRESSIONS OF THE CHICAGO SESSION

The seventy-fifth annual session of the American Medical Association which was held in Chicago, from June 9th to June 13th, was a memorable one from several points of view.

First, it was credited with the record attendance, the total registration being 7,819. The largest previous registration was 6,446 at the last meeting held in Chicago in 1908.

This session also marked an epoch in the history of the association, it being the twenty-fifth anniversary of the beginning of the organization of the medical profession through uniform county and state associations which in turn became the component parts of the great American Medical Association. Meanwhile the membership has grown until it has reached the grand total of over 90,000.

Through the genius, foresight and executive ability of Dr. George H. Simmons, this movement was started when he became editor and business manager of the Journal of the Association in 1899. The present form of organization was completed and put into operation at the St. Paul session in 1901.

And now that Dr. Simmons has lived to witness the full fruition of his plans, with the establishment of the association in its own home at 535 North Dearborn St., it was indeed a fitting tribute that on his retirement from the heavy burden of his position he was tendered a testimonial dinner at the Congress Hotel on the evening of June 9th, at which in the presence of more than 400 fellows he was presented with a life-sized portrait which will ultimately be placed in the building of the association.

This meeting was presided over by Dr. Harvey Cushing, of Boston, and addresses were made by Drs. Cushing, Frank Billings, W. J. Mayo and Mr. Will Owen Jones, editor of the Nebraska State Journal. The portrait of Dr. Simmons was presented by Dr. William S. Thayer, of Baltimore, to which Dr. Simmons responded briefly but appropriately.

The meeting place in the new municipal pier, on the lake front, was an innovation and was, in most respects, a successful experiment. The ample space available made it possible to house everything under one roof. This furnished ample space for registration, for the commercial and scientific exhibits, and also made possible the housing of the various sections of the scientific assembly close together so

that one could readily go from one section meeting to another without loss of time.

There were two serious drawbacks, however, to this arrangement. The temporary division of the space in the great pier to accommodate the various sections left much to be desired in acoustics, and the very general custom nowadays of those presenting papers to demand facilities for lantern demonstrations, if only for the exhibition of a few tables of figures, made it necessary to board the room on all sides, rendering it dark, barn-like and unattractive in appearance, and impossible of ventilation. For this last inconvenience, fortunately, the weather was cool. These disadvantages, however, were of minor importance as compared with the many advantages.

The scientific exhibit was perhaps the best and largest ever shown and there were 140 individuals who participated. An interesting feature, and one that was most popular with the visiting doctors, was the daily demonstration of fresh pathological material received from the Chicago hospitals and the Bureau of Animal Industry at the stockyards.

The opening of the general meeting was held Tuesday evening, June 10, at the Auditorium Theater, at which time President-elect Dr. William A. Pusey read his presidential address, which appeared in the Journal June 14th.

The officers elected for the ensuing year were as follows: President, Dr. William D. Haggard, of Nashville, Tenn.; Vice-President, Dr. E. B. McDaniel, of Portland, Oregon; Secretary, Dr. Olin West; Treasurer, Dr. Austin A. Hayden, of Chicago; Speaker of the House of Delegates, Dr. Frederick C. Warnshuis, of Grand Rapids, Mich.; Vice Speaker, Dr. Rock Slyster, of Wisconsin.

Two members of the Board of Trustees, Dr. Wendell C. Phillips and Dr. Frank Billings, declined to be candidates for re-election and the vacancies created by their retirement were filled by the election of Dr. J. H. Walsh, of Illinois, and Dr. Edward B. Heckel, of Pennsylvania.

Dr. Thomas McDavitt, whose term of office also expired, was unanimously re-elected.

With the retirement of Dr. George H. Simmons from active participation as editor of the Journal and general manager, there will be little change, as Dr. Fishbein, who succeeds him as editor of the Journal, has been in training for the position for some time. The same may be said of Dr. Austin A. Hayden, who is to become business manager. It is a source of satisfaction to know that the valuable services of Dr. Simmons will still be at the disposal of the association in an advisory capacity.

The meetings of the House of Delegates and of the various committees were held in the Association Building at 535 North Dearborn Ave., 132 delegates out of a total of 150 being registered. In the prompt transaction of business the house made a record and this was largely due to the ability of its speaker, Dr. Frederick Warnshuis, and the indefatigable efforts of our very able and efficient secretary, Dr. Olin West. Familiarity of the delegates with the business to come before the house also facilitated greatly the transaction of business.

State associations are coming to realize more and more the necessity of exercising great care in the selection of the delegates to represent them, and it is a common custom to return a delegate who has rendered conspicuous service, for a second or even a third term. It is a matter of record that it requires one term for the new member to become acquainted with the business before the house and to get his bearings. For the first year he is assigned no committee appointment, and perhaps only a minor one the second year, and it is the members of committees who do the work. Therefore, the new member has little more than the privilege of a seat and vote. Our policy of promoting alternates to succeed to delegates does not meet the requirements, as the alternate does not attend the meetings, except in the absence of the regularly elected delegate. It is a great honor to be selected to represent the association in the House of Delegates, second only to the presidency of the association, but instead of passing the honor along to members of our association whom we wish to recognize, would it not be well to look particularly to his qualification, his interest in the association, his familiarity with the working of the organization, and then if he makes good to continue him in office for at least a second term?

According to the recommendation of the House of Delegates, if the Board of Trustees adopts the reapportionment plan, Minnesota will have three members in the next House of Delegates.

Invitations from three cities were reported by the Board of Trustees for the next session, Atlanta, Georgia, Atlantic City and St. Paul, Minnesota. A majority of the House of Delegates expressed a preference for Atlantic City. Final choice of time and place of meeting will be decided upon by the Board of Trustees.

LYMANHURST AND PARKVIEW STAFF MEETING

The regular monthly meeting of the Lymanhurst and Parkview medical staffs will be held at the Lymanhurst School, 1800 Chicago Avenue, Minneapolis, Tuesday evening, July 22, at 7:00 o'clock.

The following program will be given:

"*X*-ray Findings and Significance of Different Types of Tuberculosis in Chests of Children," Dr. Russell Gates.

"Roentgenologic Distinction Between Tuberculous and Non-Tuberculous Diseases of the Lungs," Dr. F. S. Bissell.

"Roentgenologic Diagnosis of Gastro-Intestinal Tuberculosis," Dr. R. G. Allison.

"Roentgen Therapy in Tuberculosis," Dr. Leo Rigler.

All persons interested in tuberculosis are invited to attend these meetings and participate in the discussions.

CONFERENCE OF STATE AND PROVINCIAL HEALTH AUTHORITIES OF NORTH AMERICA

At the thirty-ninth annual meeting of the Conference of State and Provincial Health Authorities held in Lansing, Michigan, June 16 and 17, 1924, Dr. James A. Hayne, State Health Officer of South Carolina, was elected president; Dr. B. U. Richards, State Health Officer of Rhode Island, vice-president, and Dr. R. M. Olin, State Health Officer of Michigan, re-elected secretary-treasurer.

The attendance at the conference included thirty-one state health officers, one provincial (Saskatchewan) representative and a large number of guests. Following the conference proper, members and guests were given the opportunity of studying the methods of the Michigan Health Department which is housed in the State Office Building.

"The Automobile as a Public Health Hazard" was the subject of the president's address delivered at the opening session by Dr. A. J. Chesley, St. Paul.

STEARNS-BENTON MEDICAL SOCIETY

The Stearns-Benton Medical Society held its annual meeting at St Raphael's Hospital, St. Cloud, on April 17. Officers elected for the coming year are: President, Dr. C. B. Lewis, St. Cloud; vice-president, Dr. G. S. Sutton, St. Cloud; secretary-treasurer, Dr. J. N. Libert, St. Cloud.

OF GENERAL INTEREST

Dr. E. R. Crow, formerly of Green Isle, is now located in Minneapolis.

Dr. Ernest H. Morris, formerly of Austin, is now located in Canadian, Texas.

Dr. George A. Geist, of St. Paul, has announced the removal of his offices from West Seventh Street to 741 Lowry Building.

Dr. E. M. Hammes, St. Paul, has moved into new offices at 535 Lowry Building. Dr. Gordon B. Kamman is associated with Dr. Hammes.

Dr. W. H. Hengstler, who recently returned from California, is now associated in practice with Dr. C. E. Riggs in the Hamm Building, St. Paul.

Dr. Everett Charles Hartley has announced the opening of offices in the Lowry Building, Saint Paul, for the practice of obstetrics and gynecology.

Dr. W. P. Shepard, assistant director of the Students Health Service at the University of Minnesota, has been granted a year's leave of absence.

Dr. R. H. Kennicott is now associated with Drs. Frick and Smith, Los Angeles, California. They have recently moved into the new medical office building.

Announcement has been received of the marriage of Dr. Erling W. Hansen, of Minneapolis, to Miss Anna Ruth Eddy, Minneapolis, which took place June 4.

Dr. Louis A. Hauser, formerly of St. Paul, has entered private practice in New York City. He has also a teaching position on the Cornell University teaching staff.

Dr. Kenneth Phelps, Minneapolis, has received notification of his election to membership in the American Bronchoscopic Society, an organization of forty members.

Dr. L. W. Morsman, Hibbing, sailed for Europe on the steamship Volendam from New York, June 14. Dr. Morsman will spend a year doing post-graduate work in Vienna.

Appointment of Dr. Charles E. Proshek to the fellowship in obstetrics and gynecology maintained by the Swedish Hospital has been announced by the School of Medicine, University of Minnesota.

Dr. Andrew Sinamark, formerly of the Gifford Clinic, Omaha, is now associated with Dr. L. W. Morsman, of Hibbing, Minn. Dr. Sinamark limits his practice to diseases of the eye, ear, nose and throat.

Dr. Charles A. Reed, of Minneapolis, was a guest of the surgeons of the Great Northern railroad at their annual meeting in Spokane, Washington, June 23 and 24, where he read a paper on "Back Injuries."

The new deep *x*-ray therapy equipment which Dr. Edward Schons has been installing at St. Luke's Hospital, Saint Paul, is now in operation. The apparatus includes one of the first thirty-milliampere high voltage, water cooled Coolidge tubes to be installed for clinical use.

Dr. and Mrs. John F. Fulton, Saint Paul, sailed last month for England, where they are the guests of their son and daughter-in-law, Mr. and Mrs. John F. Fulton, Jr., who make their home in Oxford. Dr. Fulton will return to Saint Paul the middle of this month. Mrs. Fulton expects to spend the entire summer abroad.

Physicians from thirty-six communities were registered at the Medical Short Course at the University of Minnesota, which was recently brought to a close. Fifteen were from Minnesota, seven from Iowa, six from North Dakota, and one from Wisconsin. A second medical short course of two weeks will be conducted in September according to an announcement made by R. R. Price, director of the General Extension Division.

Public Health Summer Schools are being conducted this year for the first time by the Universities of Columbia, Michigan, Iowa and California. These courses are suggested by the United States Public Health Service and offer an opportunity to those engaged in the various branches of public health work to obtain instruction in a great variety of subjects. The National Health Council has called the attention of the profession to this new departure in University summer courses.

The New Prague Community Hospital was opened to receive patients, June 9, 1924. The hospital is to be operated by a voluntary association without capital stock, and it is provided that no profit or dividend shall ever be paid to any member or individual. It is organized and will be operated solely to provide a service to the community. The operating room is thoroughly equipped and a high grade sterilizer has been installed. There is a fully equipped separate maternity department with delivery room and excellent accommodation for patients.

The distinction of having the highest rank of any student in the past seven years belongs to Miss Huldah Thelander, of Little Falls, who was a member of the graduating class of June, 1924, at the University of Minnesota Medical School.

The leading graduate student to receive the M.D. degree is Dr. Frederick C. Eberson, who has won distinction as a graduate student of bacteriology. Dr. Eberson, who came to Minnesota two years ago, has been working under fellowships granted by the Hennepin County Medical Society.

Enlargement by approximately 100 beds of the Minnesota General Hospital, situated at the University of Minnesota, was begun June 18 when steam shovels began excavating for the Todd Memorial and George Chase Christian Memorial units on the university campus. The Todd hospital will be built especially for the treatment of eye, ear, nose, and throat cases. The Christian Memorial unit will be a cancer hospital.

Dr. L. B. Baldwin, superintendent of the University hos-

pitals, expects to have the new buildings ready for use a year from September.

A gift of $250,000 by the Citizens Aid Society of Minneapolis has made possible the erection of the cancer hospital. The Citizens Aid Society is a foundation established by the late George Chase Christian. Of the total, $200,000 will go into the building and $50,000 into special radium and X-ray equipment for treatment of cancer.

Gifts of $20,000 by Mrs. Frank C. Todd, widow of the physician after whom it is named, $20,000 by Mrs. E. C. Gale and $5,000 by Mrs. Mapes gave the nucleus of the Todd hospital fund. The regents have added from building funds money to erect a hospital costing, with equipment, about $180,000.

The capacity of the University hospitals is increased from 193 beds to approximately 300 beds by the additions.

By an act of the 1921 legislature the University hospitals are also the Minnesota general hospital, to which any county may send patients for care. In these cases half of the expense is borne by the state and half by the county.

Both hospitals will be built along Union Street Southeast and will be connected by a wing with the Elliott Memorial hospital, which stands on the river bank, overlooking East River Drive.

Approximately 95 physicians have been added to the rolls of medical practitioners in Minnesota by the University of Minnesota Medical School during the past year according to statistics in the office of Dean E. P. Lyon. Forty were graduated with the M.D. degree at the end of the fall quarter last December, two were added in April, and 53 more doctors were graduated June 18 at the close of the college year.

NEW AND NON-OFFICIAL REMEDIES

The following articles have been accepted by the Council on Pharmacy and Chemistry:

ABBOTT LABORATORIES:

Neutral Acriflavine-Abbott for Intravenous Injection, 0.1 gm. (ampules)

HOFFMANN-LA ROCHE CHEMICAL WORKS:

Digalen-Roche (Cloetta)

Ampules Digalen-Roche (Cloetta), 1.1 c.c.

Tablets Digalen-Roche (Cloetta)

Hypodermic Tablets Digalen-Roche (Cloetta)

LEDERLE ANTITOXIN LABORATORIES:

Pollen Antigens-Lederle

Giant Ragweed Pollen Antigen-Lederle; Green Sage Pollen Antigen-Lederle; Lamb's-Quarters Pollen Antigen-Lederle; Marsh Elder Pollen Antigen-Lederle; Olive Pollen Antigen-Lederle; Pasture Sage Pollen Antigen-Lederle; Southwestern Ragweed Pollen Antigen-Lederle; Western Water Hemp Pollen Antigen-Lederle; Western Ragweed Pollen Antigen-Lederle

MEAD, JOHNSON & CO.:

Mead's Powdered Protein Milk

OHIO CHEMICAL AND MFG. CO.:

Ethylene for Anesthesia

E. R. SQUIBB AND SONS:

Pollen Allergen Solutions-Squibb

Annual Salt Bush Pollen Allergen Solution-Squibb; Arizona Ash Pollen Allergen Solution-Squibb; Arizona Cottonwood Pollen Allergen Solution-Squibb; Arizona Walnut Pollen Allergen Solution-Squibb; Ash Pollen Allergen Solution-Squibb; Black Mulberry Pollen Allergen Solution-Squibb; Bermuda Grass Pollen Allergen Solution-Squibb; Black Walnut Pollen Allergen Solution-Squibb; Brome Grass Pollen Allergen Solution-Squibb; California Black Walnut Pollen Allergen Solution-Squibb; Careless Weed Pollen Allergen Solution-Squibb; Cedar Pollen Allergen Solution-Squibb; Cocklebur Pollen Allergen Solution-Squibb; Corn Pollen Allergen Solution-Squibb; Dark Leaved Mugwort Pollen Allergen Solution-Squibb; False Ragweed Pollen Allergen Solution-Squibb; Hickory Pollen Allergen Solution-Squibb; Johnson Grass Pollen Allergen Solution-Squibb; June Grass Pollen Allergen Solution-Squibb; Lamb's-Quarters Pollen Allergen Solution-Squibb; Marsh Elder Pollen Allergen Solution-Squibb; Nettle Pollen Allergen Solution-Squibb; Oak Pollen Allergen Soution-Squibb; Mugwort Pollen Allergen Solution-Squibb; Orchard Grass Pollen Allergen Solution-Squibb; Pigweed Pollen Allergen Solution-Squibb; Pine Pollen Allergen Solution-Squibb; Poplar Pollen Allergen Solution-Squibb; Ragweed Pollen Allergen Solution-Squibb; Rye Pollen Allergen Solution-Squibb; Sagebrush Pollen Allergen Solution-Squibb; Sandbur Pollen Allergen Solution-Squibb; Shadscale Pollen Allergen Solution-Squibb; Sheep Sorrel Pollen Allergen Solution-Squibb; Slender Ragweed Pollen Allergen Solution-Squibb; Sweet Vernal Grass Pollen Allergen Solution-Squibb; Timothy Pollen Allergen Solution-Squibb; Western Ragweed Pollen Allergen Solution-Squibb.

WILSON LABORATORIES:

Desiccated Parathyroid Substance-Wilson

Tablets Desiccated Parathyroid Substance-Wilson 1/20 grain

Tablets Desiccated Parathyroid Substance-Wilson 1/10 grain

NEW AND NON-OFFICIAL REMEDIES

Cryogenine Phenylsemicarbazide.—Cryogenine is an antipyretic and analgesic. It is claimed that cryogenine does not affect digestion and that it has scarcely any effect on the circulation and respiration. Cryogenine is claimed to be useful as an antipyretic in febrile conditions. As an analgesic, it is said to be of value in rheumatism, headache, sciatica, gout and in painful conditions generally. Cryogenine is marketed in the form of powder and 0.5 gm. tablets.

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Dr. Coffey has developed what constitutes the nearly ideal operation in that it controls these two factors and in addition provides for the performance of the major procedure by the abdominal route, all performed under the direct vision of the operator.

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pitals, expects to have the new buildings ready for use a year from September.

A gift of $250,000 by the Citizens Aid Society of Minneapolis has made possible the erection of the cancer hospital. The Citizens Aid Society is a foundation established by the late George Chase Christian. Of the total, $200,000 will go into the building and $50,000 into special radium and X-ray equipment for treatment of cancer.

Gifts of $20,000 by Mrs. Frank C. Todd, widow of the physician after whom it is named, $20,000 by Mrs. E. C. Gale and $5,000 by Mrs. Mapes gave the nucleus of the Todd hospital fund. The regents have added from building funds money to erect a hospital costing, with equipment, about $180 000.

The capacity of the University hospitals is increased from 193 beds to approximately 300 beds by the additions.

By an act of the 1921 legislature the University hospitals are also the Minnesota general hospital, to which any county may send patients for care. In these cases half of the expense is borne by the state and half by the county.

Both hospitals will be built along Union Street Southeast and will be connected by a wing with the Elliott Memorial hospital, which stands on the river bank, overlooking East River Drive.

Approximately 95 physicians have been added to the rolls of medical practitioners in Minnesota by the University of Minnesota Medical School during the past year according to statistics in the office of Dean E. P. Lyon. Forty were graduated with the M.D. degree at the end of the fall quarter last December, two were added in April, and 53 more doctors were graduated June 18 at the close of the college year.

NEW AND NON-OFFICIAL REMEDIES

The following articles have been accepted by the Council on Pharmacy and Chemistry:

ABBOTT LABORATORIES:

Neutral Acriflavine-Abbott for Intravenous Injection, 0.1 gm. (ampules)

HOFFMANN-LA ROCHE CHEMICAL WORKS:

Digalen-Roche (Cloetta)

Ampules Digalen-Roche (Cloetta), 1.1 c.c.

Tablets Digalen-Roche (Cloetta)

Hypodermic Tablets Digalen-Roche (Cloetta)

LEDERLE ANTITOXIN LABORATORIES:

Pollen Antigens-Lederle

Giant Ragweed Pollen Antigen-Lederle; Green Sage Pollen Antigen-Lederle; Lamb's-Quarters Pollen Antigen-Lederle; Marsh Elder Pollen Antigen-Lederle; Olive Pollen Antigen-Lederle; Pasture Sage Pollen Antigen-Lederle; Southwestern Ragweed Pollen Antigen-Lederle; Western Water Hemp Pollen Antigen-Lederle; Western Ragweed Pollen Antigen-Lederle

MEAD, JOHNSON & CO.:

Mead's Powdered Protein Milk

OHIO CHEMICAL AND MFG. CO.:

Ethylene for Anesthesia

E. R. SQUIBB AND SONS:

Pollen Allergen Solutions-Squibb

Annual Salt Bush Pollen Allergen Solution-Squibb; Arizona Ash Pollen Allergen Solution-Squibb; Arizona Cottonwood Pollen Allergen Solution-Squibb; Arizona Walnut Pollen Allergen Solution-Squibb; Ash Pollen Allergen Solution-Squibb; Black Mulberry Pollen Allergen Solution-Squibb; Bermuda Grass Pollen Allergen Solution-Squibb; Black Walnut Pollen Allergen Solution-Squibb; Brome Grass Pollen Allergen Solution-Squibb; California Black Walnut Pollen Allergen Solution-Squibb; Careless Weed Pollen Allergen Solution-Squibb; Cedar Pollen Allergen Solution-Squibb; Cocklebur Pollen Allergen Solution-Squibb; Cor... Pollen Allergen Solution-Squibb; Dark Leave... Mugwort Pollen Allergen Solution-Squibb; Fals... Ragweed Pollen Allergen Solution-Squibb; Hick... ory Pollen Allergen Solution-Squibb; Johnso... Grass Pollen Allergen Solution-Squibb; June Gras... Pollen Allergen Solution-Squibb; Lamb's-Quarter... Pollen Allergen Solution-Squibb; Marsh Elde... Pollen Allergen Solution-Squibb; Nettle Polle... Allergen Solution-Squibb; Oak Pollen Allerge... Soution-Squibb; Mugwort Pollen Allergen Solu... tion-Squibb; Orchard Grass Pollen Allergen Solu... tion-Squibb; Pigweed Pollen Allergen Solution... Squibb; Pine Pollen Allergen Solution-Squibb... Poplar Pollen Allergen Solution-Squibb; Ragwee... Pollen Allergen Solution-Squibb; Rye Pollen A... lergen Solution-Squibb; Sagebrush Pollen Aller... gen Solution-Squibb; Sandbur Pollen Allerge... Solution-Squibb; Shadscale Pollen Allergen Solu... tion-Squibb; Sheep Sorrel Pollen Allergen Solu... tion-Squibb; Slender Ragweed Pollen Allerge... Solution-Squibb; Sweet Vernal Grass Pollen Aller... gen Solution-Squibb; Timothy Pollen Allerge... Solution-Squibb; Western Ragweed Pollen Aller... gen Solution-Squibb.

WILSON LABORATORIES:

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is formed and the lower portion of the sigmoid and rectum are invaginated through the anus by traction on a rectal tube fastened to the divided end of the bowel. The peritoneal floor of the pelvis is completely closed over this dissection and by drawing in the loose peritoneum from the sides of the pelvis an extra-peritoneal cavity drainage tract (the latter formed of peritoneum) is established, draining suprapubically from the region of the base of the bladder. In the female, after closure of the peritoneum of the floor of the pelvis, a post-vaginal drainage is established.

The second stage of the operation is performed after ten days, and is a quite minor and bloodless procedure, ten or twelve minutes sufficing for its performance. In the female it is not even necessary to remove the coccyx. In the male an incision is made around the anus and extended over the length of the coccyx, the latter being removed, and the rectum and invaginated sigmoid scooped out. By placing a rubber tube down the suprapubic tract on through the operative field, with its exit through the perineum, the entire operative field in the hollow of the sacrum can be washed, kept clean, and irrigated with Carrel-Dakin fluid, thus controlling the element of sepsis, hastening healing and greatly shortening the postoperative convalescence.

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2. Dr. A. W. Collins (Duluth) gave the following case report:

C. G. D., age 34, married. Born in Maine. Occupation —executive secretary. Mother died at about the age of 70, cause unknown. Father living and in fair health, age 70 or over. No family history of tuberculosis, carcinoma, insanity or Bright's disease. Patient was a healthy child until in his teens or about twenty years ago, when he was operated upon for appendicitis, at which time the wound drained for three weeks, which he spent in bed. About one week ago, while skiing down a hill, he struck against a stone fence and hurt his right side in the neighborhood of the old operation scar and had pain along the groin and down into the right cord and testicle. Two days before I saw him, in January, about one week following the skiing accident, he made a long auto trip into the country on business and became quite chilled. He complained of much pain in the right side on that day. On Sunday, the following day, January 20, 1924, he was worse and stayed in bed. On Monday, January 21st, I saw him at the instigation of Dr. Cyril Smith. I found him lying in bed in no characteristic attitude of pain; he complained of tenderness in the right abdomen and gave me the above history, also that he had had a chill on the evening of January 19th and that he had had more or less distress in urinating.

Examination: No lung involvement. His temperature on admission to the hospital was 102, and the tenderness was exquisite over the old wound, but there was no sign of fluctuation; he was also tender out in the right lumbar region. Pressure from the back forward in this region made him wince considerably. The tenderness in the right iliac region extended upward and gave considerable spasm to the muscles in the liver region. The left rectus (muscle) was not spastic. There was no resistance in the other portions of the abdomen. On careful questioning he stated that the appendix was removed at the time of his operation twenty years ago. He insisted that the operation was done by a competent surgeon in a large city, who was chief of a surgical service, and that the appendix was removed, according to the statement of the surgeon.

The urine was scanty, very dark in color and contained no sediment. The tongue was thickly coated. The bowels had moved the day before, he having had a physic. The stereo-*x*-ray of the chest was negative. The throat was negative. On further examination of the abdomen a quick pressure over the sigmoid gave reflex pain in the region of the cecum. The white count was 19,000.

Diagnosis at that time: acute pelvic infection, possibly intestinal obstruction from old adhesions, possibly abscess in the pelvis.

Operation: Separation of intestinal adhesions and severing of constricting bands.

Operation findings: The cecum was bound down in the lumbar trough and adherent beneath the old appendectomy scar. It was folded on itself with adhesions, the last three or four inches of the ileum were pulled down into the pelvis by adhesive bands. The last two or three inches of ileum were constricted by bands. The gut was red, and, in spots, raw. The omentum at one spot adherent, red and thickened. No pus. No appendix. Adhesions separated and the constricted gut freed by cutting the bands.

Anesthetic: ether. Drainage by rubber tube.

Peritonitis developed rapidly and he died the following evening.

Autopsy showed: Spleen, adrenals, bladder, prostate, liver, stomach, small intestines, lungs, all practically negative. The external iliac glands of the right side very much enlarged, congested and edematous. Marked proliferation of the connective tissue below the old lateral appendectomy scar described above. This fibrous tissue in places very congested and edematous and shows slits filled with pus. Such spaces filled with pus are found between the fascia transversalis and the transversus abdominus, near to the iliac crest, and also between the anterior layer of the lumbodorsal fascia and the quadratus lumborum, near to the iliac crest.

The soft parts are very much congested, edematous and infiltrated around the right iliac crest, around its anterior part. The iliac crests are then cleaned of the surrounding soft parts and the anterior part of the right iliac crest is seen to be about one and one-half times as thick as the left one. On cut surface, the corticalis of the right iliac crest is thick, and very dense; the spongosa is also more dense than usual. The peritoneum at the surface of this thickened bone is also more thick and tough than on the left side. No abscess is found in the bone, no sequestrum. The only observation made is the thickening of the bone, the thickening of the periosteum, the edema and fibrosis of the surrounding tissues which are in direct connection with the above described purulent fistulous tracts. No changes can be found in the other parts of the iliac bone, in the 12th rib, or in the dorsal column.

The diameter of the thigh is the same on both sides. There are no apparent wounds of the right limb, or of the anus.

The cecum was attached to the iliac fossa somewhat tighter than usual, by tough connective tissue. (The appendix had been removed about twenty years ago and no rests in the wound of amputation could be found on the cecum.)

The periosteum of the iliac bone is not loosened at any place; there are no naked and rough bony surfaces.

No perforation of the gut at any point, the gut having been filled with water to determine this.

Anatomical Diagnosis:

Status after recent laparotomy.

Status after old appendectomy.

Retrocecal chronic inflammation.

(Reactive) periostitis of the right iliac crest.

Inflammation of the peritoneum. (Peritonitis.)

Cloudy swelling of the kidneys.

Cloudy swelling of the liver.

Chronic cholecystitis.

Smears of pus of the parietal abscesses show numerous isolated gram-positive cocci and only a few short chains of streptococci, several groups of staphylococci.

Summary: This seems to me an unusual condition of infection lurking in an old drainage wound following appendectomy, 20 years ago. The lighting up probably had its origin in the "bump" against the stone fence a week before admission to the hospital and his lack of care of himself in the interim.

The obscurity was deepened by his insistance that the appendix was removed. The doubt of this still lurked, however, in the minds of consultants and myself.

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3. Dr. A. E. Benjamin (Minneapolis) reported a case of acute appendicitis, rupture, gangrenous, and intussusception.

Mrs. W., 41, married, 2 children (20 and 10). Family history negative.

Personal history: Well, except following birth of first child had pelvic peritonitis with some pelvic trouble for some time, with pain on the left side of the lower abdomen.

Symptoms: Was taken suddenly ill on the morning of the 13th, with acute gastric disturbance and soreness in the right lower quadrant. Has had paroxysmal attacks of pain with relief between. Was sent to the hospital on the evening of the 13th. Leucocyte count 16,000; temperature 99, which rose to 100 the next morning. Urinalysis: slight trace of albumen, otherwise negative.

Morning of the 14th, patient feeling very well. Temperature 100 and pulse 100. Great deal of pain upon pressure over the appendix. No vomiting. Abdomen fairly flat.

Operation: Under local anesthetic, gas and ether, median incision was made. The left ovary was found three times normal size, fibro-cystic, with left tube enveloping it and two parovarian cysts, thin-walled and containing about an ounce of serum. The outer half of the tube and the ovary were removed, also the cysts. The gallbladder was large, but contained no stones. It was somewhat prolapsed.

The appendix was acutely inflamed and two and one-quarter inches long, not diseased at the outer half. The base of it being surrounded by inflammatory bands and the cecum creeping upon the lower third of the appendix, the base being inverted somewhat. The cecum around the base of the appendix was very much thickened and inflamed. The ileum was kinked somewhat. This whole mass was telescoped into the ascending colon, which was redundant, showing a definite beginning of intussusception. A few bands were beginning to form, holding it in this position. This was reduced as well as the base of the appendix, gently pulling out all the surrounding cecum. The appendix had ruptured at the base, but the imbedded condition prevented the pus escaping before operation. The base was entirely gangrenous as well as a part of the cecum. The appendix was removed and this portion of the cecum involved. Supplementary sutures brought the other portion of the cecum, mesentery, appendix and omentum over the involved diseased area. A Penrose drain was left to drain this diseased area.

Patient operated upon this morning, May 14, 1924.

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4. Dr. A. Schwyzer (St. Paul) gave an additional report of a case:

This case was presented before the Academy two and one-half years previously. Patient, a lady, then 69½ years old, was operated on two and one-half years ago. Cholecystectomy was done for severe cholecystitis with stones; and because there was great thickening in the cystic duct we inadvertently removed the whole of the hepatic duct. This case was then published last fall in the "Surgical Clinics of North America." We were not sure at the time of operation that we had injured the duct, but thought we had, because there was a suspicious looking cord running upward. While the assistant closed up the wound I went into the laboratory and cut the specimen open. In the wall of the cystic duct we noticed a little tube. A frozen microscopic section showed epithelium. The shape of the tube showed that the upper end was near the bifurcation. We had not only removed part of the hepatic duct, but had removed every bit of it. The wound was amply drained. I went in again after six days, which I thought would be just long enough to have the effect of the operation overcome and short enough so that I could still open the wound easily. We found, back of the duodenum and covered by fibrin, the tied-off common duct. On the liver there was the raw surface from the cholecystectomy and no sign of the duct. However, we found a little catgut on a small cord and hoped that was where we tied the duct. We loosened that ligature and it started to pump; it was the cystic artery. No place could be made out on the surface of the liver where the duct should be. But mopping carefully we finally saw a little oozing of bile, so we took a large uterine sound and went in. It slipped in easily toward the left, so that we knew we were in the left hepatic duct. Some bile escaped along this sound. We introduced a small catheter cut on a slant and left it there. The stump of this cystic artery was used to anchor it with chromic catgut. The lower part of the tube was also cut on a slant so as not to cause any pressure. The catheter was left long, so as to get the lower end out of and beyond the duodenum. After dilating the papilla, the tube went in easily. We could readily approximate the upper end of the common duct to the liver, but there was nothing to hold it except that cystic artery stump.

The tube was left in till now. It was thus in for two and one-half years, and the patient came to town at different intervals. Each time the *x*-ray picture was identical, with the tube remaining in place. She was perfectly well. I had word from her two years after the operation that she did her work and was well.

All went well until about three weeks ago, when she developed chills, temperature 103 degrees to 103½, and

sne became severely jaundiced. She was brought to the city. We put her on Carlsbad, hoping for a gradual decrease of the fever. At the end of 8 days the same condition existed. From the *x*-ray picture it appeared that the tube was still in place. We had to remove that tube. But, perhaps, had the tube come out of the duct and was the present condition due to a stricture? When the tube was removed by nicking the duodenum and grasping the tube with a thin artery forceps, it retained the shape it shows in the *x*-ray picture, and was quite stiff. You see here the tube. I throw it on the table and it retains the same curves which you saw in the *x*-ray two and one-half years ago and which you see in the one taken immediately before this last operation. That is undoubtedly the reason why it was not passed.

In the upper opening of the tube we noticed a smeary greyish-looking material. We figured if this was pus, the tube had remained in place, as otherwise food would have washed it clean of pus. If the tube had been in place, our operation promised a good result. We thus made a smear of this material and found it to be pus with an immense amount of bacilli of hay-bacillus shape.

The patient thus had an infectious cholangitis. The chills stopped and now, after eight days, she is free from fever, sits up, and the jaundice has already nearly disappeared.

Dr. Schwyzer also showed autopsy specimen of a case of aneurysm of the uppermost part of the descending aorta, perforating in three places into the left bronchus. Case died very suddenly from hemorrhage. The posterior wall of the aneurysm had disappeared from pressure against the vertebral column. Three vertebræ had been eroded with the intervertebral cartilaginous discs protruding, as they are less readily yielding to pressure.

Dr. S. E. Sweitzer (Minneapolis) then gave his inaugural thesis, entitled "Protean Skin Diseases, Including Syphilis; Lantern Slide Demonstration."

DISCUSSION

Dr. C. D. Freeman (St. Paul): The method by which Dr. Sweitzer has made his entré into the Academy of Medicine is very commendable. When I heard he was to give a lantern slide demonstration instead of a thesis on some rare skin disease, I told him he used good judgment. To the average physician dermatology is a vague subject and an ultra-scientific paper by him before a body of general men would be similar to a surgeon, neurologist, or oculist delving into the intricacies of their specialty before a dermatological society.

He has practically made a clinical evening of it and has shown slides of cases many of you can recognize. I think Dr. Sweitzer is to be congratulated on his collection.

Dr. Paul Cook (St. Paul): I only wish to add that the change in the last twenty years has been very marked in the teaching of dermatological subjects. With the development of photography and showing of lantern slides, I think all physicians will be better trained in dermatology. Heretofore the ordinary courses in dermatology have consisted of lectures. The only way one can teach dermatology is to show patients with the disease or show lantern slides that will picture it clearly. I think in a few years opticians will develop lenses so that they will show the lesions better, and there are great possibilities in color photography. Heretofore one had to go into the clinic and spend an immense amount of time examining patients.

The meeting adjourned.

John E. Hynes, M.D.,
Secretary.

PROGRESS

Abstracts to be submitted to Section Supervisors.

MEDICINE

SUPERVISORS:
F. J. HIRSCHBOECK,
FIDELITY BLDG., DULUTH
THOMAS A. PEPPARD,
LA SALLE BLDG., MINNEAPOLIS

DISTURBANCES OF RENAL FUNCTION IN PERNICIOUS ANEMIA: Edward J. Stieclitz (Arch. of Int. Med. for Jan. 15, 1924) points out that in pernicious anemia there is frequently evidence of mild renal damage, manifesting itself by albumin, casts, or both, in the urine. He brings out the fact that up to the present time it has been assumed that this renal irritation or damage is the result of malnutrition and anoxemia of the kidneys, due to the anemia. However, this is by no means as pronounced in cases of secondary anemia. While admitting that the hemolytic intoxication of pernicious anemia may injure the kidneys directly, he brings out the fact that the deposition of iron in the kidneys may cause mild nephritic changes in experimental animals, and that therefore renal hemosiderosis might be responsible for the renal disturbances in pernicious anemia.

With regard to the incidence of renal disturbance, Cabot has reported an incidence of 46 per cent albuminuria in 506 cases of pernicious anemia, whereas at Johns Hopkins, in 50 cases, the incidence was 62 per cent. On the other hand, in 100 cases of secondary anemia, associated with a great variety of diseases, from the Presbyterian Hospital, Chicago, albuminuria was present in only 14 per cent. This shows a very striking difference in the occurrence of albuminuria in the two types of anemia.

The relationship between the hemoglobin content of the blood and the presence or absence of renal changes is interesting. Out of 150 cases of pernicious anemia, the average hemoglobin content was 41 per cent; the average of the albuminuric cases was 36 per cent, and for those without albumin 46 per cent.

The observations of many men as to the clinical character of the renal disturbance show that there is rather fixed low urinary specific gravity in connection with this disease, and that this is more pronounced with the decrease in the hemoglobin content of the blood. The PSP test usually shows a good output; blood chemistry usually normal, except for a slight increase in the uric acid content.

In regard to the pathologic changes, he states that these have not been as clear as the clinical changes. The study of the kidneys at necropsy has not yielded evidence of

uniform pathologic changes. However, from the protocols from the pathologic laboratory of Johns-Hopkins hospital of necropsies of 36 cases of pernicious anemia, 83 per cent of these showed renal pathologic changes. The most common and uniform finding was tubular degeneration, which occurred in 53 per cent of the cases. In 42 per cent there was a gross renal hemosiderosis. Where special study was made, microscopic hemosiderosis was found in 14 out of 15 cases.

His conclusions are that the most characteristic change in the kidney of pernicious anemia is perenchymatous tubular degeneration, whereas the second, and probably the more important, finding is the renal hemosiderosis.

Experimental studies by several men have demonstrated the fact that the iron content of various organs was definitely increased in pernicious anemia. This relative increase is most marked in the kidneys, but also very evident in the liver and spleen. It has been shown that the deposition of iron in the kidneys is practically constant in its distribution, and exists as a granular deposit in the cells of the convoluted tubules, proximals and distals. Occasionally it is also found in the cells of Henle's loop. Many observers have reported that there was no iron to be demonstrated in the glomerular space or in Bowman's capsule. The same distribution has been shown through experimental production of renal hemosiderosis. There is a great uniformity of the pathological and experimental reports concerning this. The convoluted tubule cells are almost universally conceded as being the site of secretion and deposition of iron-containing pigment or simple iron salts. It has been shown that the cases showing the largest amount of iron in the tubules had a lower hemoglobin reading at the time of death than the others. The author states that this may be explained in one of two ways: (1) Either that the anemia is the direct cause of the renal disturbance, or, (2) that at the time, the renal siderosis is most marked, and therefore the effects of the iron most conspicuous.

Experimental work on dogs, which is reported in the article, shows that simple secondary anemia alone does not cause any conspicuous change in renal function or in kidney damage, as manifested by albumin and casts, whereas an anemia with hemoglobinemia, and therefore renal hemosiderosis, leads to distinct renal damage, as evidenced by the fall in the phenolsulphonphthalein excretion and the appearance of albuminuria and casts.

Experimentation has also shown that iron, during its elimination by the kidneys, causes a fall in the rate of water excretion, and a fall in the specific gravity, indicating a diminution of all secretion at that time.

He summarizes his observations and results as follows: "It has been shown that:

(1) Disturbances of renal function are common in pernicious anemia.

(2) Histologic examination of the kidneys frequently discloses degeneration of the tubular epithelium.

(3) There is a deposition of iron in the convoluted tubules of the kidneys.

(4) Iron in such a location probably causes damage to the epithelium.

(5) Iron in these cells inhibits the passage of water and total solids, and reduces the specific gravity of the urine.

(6) Fixation of the specific gravity and an increase of the night over the day urine volume is characteristic of the alteration of renal function in pernicious anemia. The correlation of these findings leads to the conclusion that the renal functional changes in pernicious anemia may be attributed, at least in part, to the accumulation of iron in the convoluted tubules, rather than to the anemia alone. The hemosiderosis and the resulting changes in function are so specific that one may speak of this condition as a nephritis of pernicious anemia. It is particularly interesting in view of the fact that the changes are apparently purely tubular, and result from a perverted metabolism."

P. G. Boman.

THE CORRELATION OF AUTOPSIES AND CLINICAL MEDICINE IN TUBERCULOSIS: W. B. Jameson (American Review of Tuberculosis, April, 1924). The writer reports 100 autopsies made at the Hamburg (Pennsylvania) State Sanatorium for Tuberculosis.

Ten cases had not shown clinical tuberculosis. One abdominal mass diagnosed as tuberculous proved to be carcinoma with metastasis to lymph nodes. Another patient had an old mitral heart lesion and induration of the liver, spleen and kidneys. Several others showed emphysema and other changes due to inhaled dust in the coal mines. Of the 90 patients with clinical tuberculosis 28 had pneumonokoniosis and emphysema, but all cases of lung tuberculosis had emphysema of the air vesicles immediately surrounding the tuberculous masses. The right heart showed changes in the cases of advanced tuberculosis. A portion of the lung is destroyed by cavitation, fibrosis or tuberculous masses and emphysema weakens the usefulness of the rest and a severe strain is put on the heart. Almost all the hearts of this autopsy series showed atrophy and there was always dilatation of the right auricle, ventricle and tricuspid opening and hypertrophy of the right ventricular wall. Again with the inability of the heart to perform its function there was general congestion with cyanotic induration of liver, spleen and kidneys. The size of the heart in this series was not reduced and averaged about 1 to 155 lbs. per body weight as compared with 1 to 162 lbs. or more as given by Piersol's Anatomy for normal hearts. Seventy-three of the 90 cases showed tuberculosis in all five lobes. In only 3 cases was it limited to one lobe. Nearly all had some grade of cavitation. Most of the cavities did not have fibrous limiting membranes. In patients whose condition had been quiescent a year or more, when activity developed and death ensued, the common post-mortem findings were lungs fibrosed at one or both apices with sharply limited cavitation and nearly all the remaining lung tissue studded with tuberculous masses varying in size from one-eighth to a half inch in diameter.

Following a hemorrhage, if there is a persistent elevation of temperature and death ensues, the autopsy is prone to show widely distributed masses of such tuberculous tissue.

The extension of lesions which occurs ordinarily a few months before death involves usually all the lobes and nearly all the vesicular lung tissue. It is surprising how small an amount of air containing lung tissue can support life. Some chests showed only a portion of one lower lobe capable of respiratory function.

In three cases of fatal hemorrhage the source of the

bleeding was as follows: In the first a wide open vessel about one-eighth of an inch in diameter was found in the wall of a cavity near the floor; in the second an aneurysm of a vessel traversing a cavity had ruptured; in the third a globular aneurysm rather more than a quarter of an inch with laminated wall and ragged opening was found.

Spontaneous pneumothorax was found in thirteen cases.

Every case showed chronic fibrous adhesive pleurisy usually greater in the posterior upper region. Sometimes its thickness exceeded one-quarter of an inch. Frequently the entire pleural cavity was eliminated.

None of the patients died soon after the pneumothorax in cases where it was recognized before autopsy.

One case of pneumothorax communicated with a lung cavity.

At 35 of the autopsies typical tuberculous ulcers were seen in the small intestines. Thirty-two showed characteristic ulcers in the colon, and in all the cases there was tuberculosis of the appendix.

One case had a 12-ounce amyloid spleen; 19 showed amyloid changes in spleen; 11 in the liver and 4 in the kidney. An enlarged painless liver is usually amyloid in cases of advanced tuberculosis. Fifty-one gall bladders showed abnormal adhesions to adjacent structures.

In all cases of tuberculosis of the intestinal tract the corresponding lymph nodes were enlarged. The peribronchial and mediastinal nodes were always enlarged but almost never softened.

ARTHUR T. LAIRD.

MERCURIC CHLORIDE POISONING: H. B. Weiss (The Arch. of Int. Med., Feb. 15, 1924), in discussing this subject, has given a very definite outline of the treatment that should be followed in the various phases seen in these cases.

Mercuric chloride poisoning cases are seen not infrequently, and the vast majority of these are among young women who have taken this poison with suicidal intent.

When this poison enters the system it is very quickly taken up, and it has been shown in his experimental animals that the mercury has been detected in the blood three minutes after a tablet was placed in the animal's stomach, i.e., when the dose of the mercuric chloride was .5 grm. per kilogram of body weight. In the smaller doses the time required for detecting it was longer.

From this it is evident that there are not any of the so-called specifics which would be of avail, and that there is nothing which can be used which would combine with the mercury to produce a non-toxic subject which can be eliminated. The action of the mercury is mainly degenerative in character, and affects especially the kidneys. The degree of degenerative changes occurring will depend upon the dosage and the duration of the intoxication. Death results from the kidney injury, although all the organs of the body are affected.

Weiss mentions that one of the most constant findings after bichloride poisoning is an acid intoxication, as evidenced by a reduction of the alkali reserve. There is also a diminution of the concentration of the whole blood chloride. You have a retention of the nitrogenous elements, such as urea nitrogen, uric acid and creatinine.

The urinary findings are significant. Albumin appears early. Soon there is a diminution in the quantity of urine, and this is often the forerunner of a complete anuria. As the quantity of urine diminishes, the organic elements increase, and casts, blood, pus cells, are frequently found. In severe cases the urine is often bloody. The most constant finding has been a highly acid urine. As the intoxication progresses all the signs of uremia may be present. Headache, coma, convulsions, are not infrequent. Generalized edema may occur.

In discussing the treatment he emphasizes the fact that it is exceedingly difficult, and that there are no so-called "specifics." The problem is how to overcome the effects of the poison, and that it is useless to attempt to counteract the metallic poison itself. The logical treatment appears then to be:

(1) To prevent the progress of the pathologic changes.

(2) To eliminate the toxic material as soon as possible.

The treatment is divided into several stages:

(a) As soon as the patient comes under observation a stomach tube is introduced, and the stomach is washed with 2 qts. of a saturated solution of sodium bicarbonate. This is continued until the washings return clear.

Before removing the tube, 6 oz. of saturated solution of magnesium sulphate are introduced and allowed to remain in the stomach.

A soapsuds enema is then given.

(b) To introduce alkalines in the system the patient should receive an intravenous injection of alkali as soon after the preliminary treatment as possible. Fischer's solution has been used. This consists of crystallized sodium bicarbonate (NA_2CO_3—10H20), 10 grms., and sodium chloride 15 grms., both disolved in 1000 c.c. of distilled water. If this is not available, a solution of 4 per cent sodium bicarbonate may be used. Ordinarily, 1 to 1½ liters can be tolerated without any difficulty. The alkaline therapy should be continued by mouth. The method which he uses for this consists in dissolving 4 grms., or 1 teaspoonful, of potassium bitartrate, in 2 grms., or ½ teaspoonful, of sodium citrate, in a glass of water, orangeade or lemonade. The patient receives 8 oz. of this drink from six to eight times a day from the onset. Hot packs have not been used, except in a few patients who had a complete suppression of urine.

A liberal diet is allowed, including meat, after the diarrhea ceases.

The treatment is controlled by urinalysis, and an effort should be made to keep the urine alkaline to methylene red, and keep it so.

Weiss has treated a series of 135 consecutive patients, with a mortality of less than 6 per cent. He states that the vast majority of the patients placed on this treatment do not develop any evidence of serious kidney disease except for a moderate albuminuria, which rapidly clears. A free secretion of urine is maintained. The patient usually makes a clinical recovery in from twelve to fifteen days. Examination of several of his patients a year after they were poisoned and treated, did not show any abnormality which could be connected with the poisoning.

P. G. BOMAN.

SURGERY

SUPERVISORS:
DONALD K. BACON,
LOWRY BLDG., ST. PAUL
VERNE C. HUNT,
MAYO CLINIC, ROCHESTER

CHILLS OCCURRING EARLY IN APPENDICITIS BEFORE OPERATION AND THEIR INDICATION OF AN OPERABLE STAGE OF PYLEPHLEBITIS: William Thalhimer (Arch. Surg., Mar., 1924). Thalhimer believes that single or repeated chills during the course of appendicitis usually denote septic thrombus formation in the branches of the portal vein. Pylephlebitis is well recognized as a postoperative complication and the author states that it may begin before operation, a chill usually accompanying the onset. If allowed to progress it gradually extends throughout the portal system and sets up multiple abscesses in the liver. These may in turn infect and cause thrombosis in the hepatic veins. From this point the process extends to the lungs, sets up pulmonary abscesses and progresses to fatal termination. In the early stages, where the process is confined to the portal system, the blood culture is negative; later, after invasion of the hepatic veins, it becomes positive.

Thrombosis may occur with any intra-abdominal focus of sepsis, as ulcerative colitis, sigmoid diverticulitis, etc., but is most common following appendicitis. It is most frequently found with the red edematous turgid type, in which the meso-appendix is also thick and edematous and should be looked for in the small radicles of the ileo-colic vein, if necessary nicking the veins with a knife to determine presence or absence of bleeding. If thrombosis is found, the ileo-colic vein should be ligated at a point beyond the thrombosed area and the diseased portion excised. The author cites three cases in which the above described procedure was followed with satisfactory results and a fourth in which permission for a secondary operation was refused which terminated fatally.

DONALD K. BACON.

PEDIATRICS

SUPERVISORS:
CHESTER A. STEWART,
LA SALLE BLDG., MINNEAPOLIS
ROY N. ANDREWS,
MANKATO CLINIC, MANKATO

CONGENITAL SYPHILIS: PREVENTION AND TREATMENT: Stewart H. Welch (Arch. of Ped., Feb., 1924). Early invasion of the fetus by the spirochete pallida terminates in destruction of the fetus in almost every instance. Infection of the fetus after the middle of pregnancy may terminate in death. On the other hand, it is the latter half of gestation which furnishes the living babies who are infected. In reviewing the pathology of the fetus, we find that the placenta becomes the primary focus, and that the organisms are carried to the fetus, not as in acquired syphilis, through the lymphatic system, but in the blood stream. This permits us to separate congenital syphilis from acquired syphilis and to define the former as a maternal infection of the placenta and a transmission of this infection from the primary focus to the fetus by the blood stream. This in contradistinction to the acquired form, or an infection of the primary site which becomes the focus, and the general transmission through the lymphatic system. Proper supervision is just as much a part of the treatment as is mercury. No congenital clinic is complete nor can it promise much success unless a competent nursing staff is available for follow-up visits and to maintain regularity in attendance for treatments. All standard methods of treatment have their applications. The truly specific drug is mercury. Administration by mouth given by parent or foster nurse over a period of two or three years is almost in every instance uneventful, incomplete or otherwise unsuccessful. Inunctions are filthy labels of the malady; a mother soon wearies of her irksome task, and after a short time you can feel sure she will develop inefficiency. Absorption of mercury by the skin is influenced by too wide individual variations. The only satisfactory therapy is the administration of the specific drug by the physician at regularly selected intervals. This permits frequent pediatric check as to the child's general condition, including diet, stools, and weight, and a recorded definite amount and interval of dosage, and safeguards intolerances. Any method which permits quick control, maintains the control, and tends to decrease the duration of needed administration is of inestimable value. With this conviction, the method of choice is the giving of the selected mercurial and arsphenamin intramuscularly or intravenously, and iodides when needed as an adjunct by mouth.

R. N. ANDREWS.

RONTGENOLOGISCHE STUDIEN UBER DIE PERISTOLISCHE FUNKTION DER MAGEN IM SAUGLINGSALTER UND IHRE BEDENTUNG FUR DIE ENTSTEHUNG DES HABITUELLEN ERBRECHENS: Julius Rogatz (Zeitschr. f. Kinderheil., Bd. xxxviii, s. 1-11). Rogatz defines the peristolic function of the stomach as the ability of the organ to concentrically surround and to adapt itself to the volume of its contents. In an interesting and valuable paper, he points out that this peristolic function of the stomach is present in infants, also. It is present, however, only after feeding of thick cereals. He found that a thick feeding of flour and milk became rapidly fluid in the stomach while thick potato and vegetable feedings retained their consistency in the stomach. From a series of *x*-ray studies of the stomach he points out that after thick cereal feedings the stomach assumes a round or oval form and is reduced to a third of its former size (i.e., when empty). The value of thick cereal feeding to habitual vomiters or babies with pylorospasm is thus placed on a more rational and scientific basis.

DAVID SIPERSTEIN.

BOOK REVIEWS

BOOKS RECEIVED FOR REVIEW

APPLIED PATHOLOGY IN DISEASES OF THE NOSE, THROAT AND EAR. By Joseph C. Beck, M.D., F.A.C.S. St. Louis: C. V. Mosby Co., 1923. $7.50.

As is evidenced by the title of this monograph, the author has placed before the specialist a definite knowledge of pathologic changes occurring within the respective organs.

The book cannot be considered a textbook (in fact, the author so informs us), as the contents are based entirely upon the author's personal experiences. Treatment is advocated on the basis of the respective pathologic conditions present. The book is adapted both for the general practtitioner and specialist, perhaps appealing more to the latter for its concise and explicit manner of placing the subject matter before the reader.

The book is profusely illustrated and from the bookmaker's art has been well put together.

GEORGE C. DITTMAN, M.D.

MANAGEMENT OF DIABETES. George A. Harrop, Jr., M.D., Associate in Medicine, College of Physicians and Surgeons, Columbia University, and Assistant Visiting Physician, Presbyterian Hospital, New York. Introducton by Walter W. Palmer, M.D. 190 pp. New York: Paul B. Hoeber, 1924. Cloth, $2.00.

HOSPITAL ORGANIZATION AND OPERATION. Frank E. Chapman, Director Mt. Sinai Hospital of Cleveland. 285 pp. Illus. New York: The Macmillan Co., 1924. Cloth.

FIRST STEPS IN ORGANIZING A HOSPITAL. Joseph J. Weber, M.A., Editor *The Modern Hospital*; formerly executive secretary Committee on Hospitals, State Charities Aid Assn. of New York; Associate Director, Boston Dispensary. 220 pp. Illus. New York: The Macmillan Co., 1924. Cloth.

COMMON DISORDERS OF DIGESTION. John L. Kantor, Ph.D., M.D., Chief in Gastro-intestinal Diseases, Vanderbilt Clinic, Columbia University; Associate Gastroenterologist and Associate Roentgenologist, Montefiore Hospital for Chronic Diseases, New York City. 245 pp. Illus. St. Louis: C. V. Mosby Co., 1924. Cloth, $4.75.

THE SCIENCE AND ART OF ANESTHESIA. Col. Wm. Webster, D.S.O., M.D., C.M., Professor of Anesthesiology, University of Manitoba Medical School; Chief Anesthetist, Winnipeg General Hospital, etc. 214 pp. Illus. St. Louis: C. V. Mosby Co., 1924. Cloth, $4.75.

DIABETES. Philip Horowitz, M.D. 231 pp.; 34 text illus.; 2 color plates. Second edition, revised and enlarged. Cloth, $2.00. New York: Paul B. Hoeber, 1924.

GASTRIC AND DUODENAL ULCER. Sir Berkeley Moynihan, Leeds, Eng. 48 pp. 2/9 post free. Bristol, Eng.: John Wright & Sons, 1923.
